American Journal of Diseases of Children

VOLUME 46 AUGUST, 1933 NUMBER 2

LACTIC ACID OF SPINAL FLUID IN MENINGITIS

PRACTICAL DIAGNOSTIC AND PROGNOSTIC VALUE

ADOLPH G. DE SANCTIS, M.D.

JOHN A. KILLIAN, PH.D.

AND

TERESA GARCIA, M.A.

NEW YORK

In 1926,[1] one of us (Dr. Killian) and his associate reported that spinal fluid from cases of meningitis showed unusually high values for lactic acid and low ones for sugar. The source of the increased lactic acid appeared to lie in the metabolic activity of the leukocytes. These studies were continued, and in 1928 [2] we came to the conclusion that the changes in lactic acid concentration were a better index of the progress of the infection than the changes in the sugar content. In meningitis the lactic acid of the spinal fluid was greater than that of the blood, but the sugar of the spinal fluid was less than that of the blood. Furthermore, the lactic acid of the spinal fluid may fluctuate within a wide range, although the amount of sugar remains normal. Since our first observations, similar studies have been reported by others.[3] In the recent paper by Wright, Herr and Paul [4] on the spinal fluid in suppurative meningitis, an excellent review on this subject is found, and it is therefore unnecessary to repeat such a summary here.

In the present report we wish to add further evidence to support the original conclusions and to enlarge on their significance. An intensive study of the sugar, lactic acid and cell count of the spinal fluid in various types of meningitis was made in order to determine whether the lactic acid varies with the cell count and whether it may be utilized as an index of the progress of the infection or of the

From the Departments of Pediatrics and Biochemistry, New York Post Graduate Medical School of Columbia University.

1. Osnato, M., and Killian, J. A.: Significant Chemical Changes in the Spinal Fluid in Meningitis, Arch. Neurol. & Psychiat. **15**:738 (June) 1926. Killian, J. A.: Proc. Soc. Exper. Biol. & Med. **23**:255, 1926.

2. Garcia, T.; Killian, J. A., and De Sanctis, A.: The Lactic Acid and the Sugar Content of the Spinal Fluid in Meningitis, Arch. Path. **6**:530 (Sept.) 1928.

3. Nishida, K.: Orient. J. Dis. Infants **4**:25, 1928. Geldrich, J.: Jahrb. f. Kinderh. **124**:159, 1929. Scheller, R.: München. med. Wchnschr. **73**:1652, 1926.

4. Wright, S. I.; Herr, E. F., and Paul, J. R.: J. Clin. Investigation **9**:443, 1931.

TABLE 1.—*Observations in Cases of Meningococcic Meningitis, with Death*

Patient	Age, Years	Sex	Date	Spinal Fluid: Lactic Acid, Mg. per 100 Cc.	Spinal Fluid: Sugar, Mg. per 100 Cc.	Spinal Fluid: Total Leukocytes per Cc.	Date of Death	Comment
V. C.	5 11/12	M	1/28	144.2	Trace	2,599	1/29	
F. di G.	1 8/12	M	6/17	97.7	0	.....	7/ 2	6/17: blood sugar, 111; lactic acid, 22.7 mg. per 100 cc.
			18	121.6	0	1,960		
			22	72.5	0	360		
			29	108.9	0			
			7/ 2	108.0	0			
H. S.	2 11/12	M	3/30	108.3	0	41,400	3/30	
				119.0	27			
D. S.	4	M	2/26	177.0	0	160,000	2/28	
			27	162.5	0	45,400	....	2/27: spinal fluid-protein, 1,120
				142.9	5			
			28	121.5	61	.....	....	2/28: spinal fluid-protein, 2,345 mg. per 100 cc.
				148.7	0			
V. D.	5 4/12	M	4/10	146.8	0	9,200	4/19	4/10: blood lactic-acid, 45.4 mg. per 100 cc.
				152.7	7			
			11	112.1	0	8,911		
				111.5	0			
			12	114.0	0	16,155		
			14	205.7	13.5	5,500		
E. B.	5 10/12	F	3/ 6	154.4	0	4,830	3/ 9	
			8	98.3	0	7,320	....	3/8: spinal fluid-protein, 714 mg.
				112.7	0			
				98.9	0			
				135.4	0			
			9	115.3	0	51,500		
E. K.	4 10/12	M	8/ 2	172.9	4	8,800	8/14	Meningococcic meningitis and pneumonia
			3	141.1	0	50,000		
			4	78.8	58	6,200		
			6	105.5	0	2,180		
			8	109.6	0	28,908		
			9	78.8	7	8,280		
			10	.....	..	5,000		
			12	95.1	45			
			13	90.4	27	1,290		
			14	79.4	0	990		
A. J.	8 3/12	F	4/ 4	149.3	0	890	4/15	
			5	156.9	0	34,666		
				135.4	27			
			6	111.5	0	46,680		
R. S.	3 3/12	F	3/25	114.3	0	20,840	4/19	
			26	93.9	4	2,800		
			27	78.1	24	1,200		
				84.4	0			
				86.9	0			
			29	49.8	31	642		
			30	52.6	18	6,100		
			31	51.7	38	852		
			4/ 2	56.7	19	970		
				54.2	21			
			3	41.6	0	299		
			6	50.4	66	2,240		
			8	42.8	21	644		
			14	44.1	28	39	....	4/14: blood sugar, 74; lactic acid, 59.9 mg. per 100 cc.
				52.3	28			
			16	63.6	42	276		
			17	67.4	27	100		
			18	65.5	18			
L. A.	2 7/12	M	3/25	119.7	0	1,575	4/ 2	
			26	116.5	39	7,800		
			27	139.9	4	4,400		
					0			
					25			
			28	129.1	115	2,200		
			29	96.4	67	580		
				75.0	103			

TABLE 2.—*Cases of Meningococcic Meningitis, with Cure*

Patient	Age	Sex	Date	Spinal Fluid: Lactic Acid, Mg. per 100 Cc.	Spinal Fluid: Sugar, Mg. per 100 Cc.	Spinal Fluid: Total Leukocytes per Cc.	Comment
H. P.	17	M	11/19	119.7	7	15,840	11/23: p_H, 7.62; protein, 108.7 mg.; chlorides, 703 mg. per 100 cc.; 11/26: protein, 85.2; chlorides, 709; 12/17: protein, 235 mg. per 100 cc.
			20	115.8	0	9,600	
			21	71.8	19	2,080	
			22	48.5	27	1,920	
			30	52.9	37	640	
			12/ 8	39.1	25	580	
			10	97.0	4	2,240	
			11	61.7	8	.....	
			26	28.4	7	1,250	
M. B.	7	F	4/27	41.0	79	1,520	
			28	47.3	55	.....	
			30	51.0	48	.....	
			5/ 2	32.1	64	222	
			3	51.7	30	500	
			5	40.6	27	650	
			6	42.2	33	1,282	
			7	35.6	41	394	5/7: protein, 121 mg. per 100 cc.
			8	64.3	21	634	
			9	62.1	45	400	
			10	67.4	28	677	
			12	39.7	64	68	
			17	42.8	64	100	
E. McT.	3 9/12	F	4/18	128.8	0	.....	4/18: blood sugar, 106 mg.; lactic acid, 29.9 mg. per 100 cc.
			19	88.8	30	4,422	
			25	27.7	60	904	
			26	27.4	55	100	
			27	24.9	50	261	
			28	28.4	42	238	
			29	91.7	21	.....	
			30	47.6	64	367	
			5/ 1	35.3	34	1,758	
			2	34.3	45	.....	
			3	35.3	37	.....	
			4	34.3	31	.....	
			5	25.9	30	.....	
			9	41.6	33	.....	
			10	46.6	8	.....	
			15	22.7	39	.....	
L. C.	9 6/12	M	8/23	92.3	0	3,830	
			24	102.7	0	3,500	
			25	74.7	8	1,160	
			26	67.1	22	870	
			27	63.6	Trace	810	
				49.1	Trace	.....	
C. G.	.....	M	9/24	75.6	31	.....	
			25	48.5	64	3,900	
			27	41.6	61	950	
			28	44.1	48	480	
			29	36.5	60	900	
D. S.	1 8/12	M	4/14	122.2	7	6,500	
			15	51.7	66	5,044	
			16	35.0	45	4,444	
			17	28.4	52	1,200	
			18	26.5	48	.....	
			19	26.5	39	600	
			20	25.2	41	206	
			24	25.8	41	3,040	
			26	25.8	45	167	
			27	27.1	48	131	
			28	34.0	45	.....	
			5/ 2	29.6	52	157	
M. S.	2 6/12	M	4/29	91.3	28	.....	
			30	51.0	61	4,600	
			5/ 1	48.5	58	2,133	
			2	....	57	1,048	
			3	31.5	57	176	
			4	27.4	54	.....	
			5	29.0	48	2,910	
			6	28.4	44	62	
			11	63.6	5	1,100	
			13	69.9	4	.....	
			14	79.4	0	274	
			18	69.3	0	1,800	
			20	68.0	0	1,800	
			23	53.2	31	550	
			6/ 2	34.7	..	.....	

TABLE 2.—*Cases of Meningococcic Meningitis, with Cure—Continued*

Patient	Age	Sex	Date	Spinal Fluid: Lactic Acid, Mg. per 100 Cc.	Spinal Fluid: Sugar, Mg. per 100 Cc.	Spinal Fluid: Total Leukocytes per Cc.	Comment
L. F.	9 11/12	F	11/17	....	0	6,120	
				56.8	28	.....	
			18	39.6	32	3,250	
			19	40.1	44	940	
			20	40.1	57	480	
C. S.	3	F	11/16	69.7	25	2,490	
			17	68.0	48	2,180	
			18	55.5	48	1,300	
			19	45.4	55	.....	
B. P.	10/12	M	9/16	52.0	38	675	
			17	32.4	47	1,016	
			18	35.3	39	1,230	
			19	55.1	50	640	
			20	33.4	50	230	
			21	35.9	55	960	
			24	52.0	55	95	
R. V.	7	F	3/13	79.4	50	7,200	
				77.5	28	.....	
			14	....	44	1,856	
					60		
			18	37.8	39	6	
			22	72.2	0	630	
			23	70.6	11	1,600	
			24	49.8	45	3,400	
			26	55.4	28	240	
			27	81.9	8	1,170	
				51.0	11		
				58.0	4		
			28	58.6	34	99	
			29	40.3	38	83	
			30	30.2	50	130	
			31	43.5	19	110	
			4/ 2	35.3	45	42	
			3	26.5	41	10	
			7	29.6	41	.....	
			13	41.0	79	.....	
S. K.	10	M	10/24	138.6	0	5,800	
			26	50.1	55	780	
			27	53.9	39	1,360	
M. P.	10	F	5/10	164.4	0	27,933	
				122.2	0	.....	
			11	74.3	13	2,444	
			12	76.2	8	765	
			13	65.2	36	.....	
			14	65.5	4	263	
			20	91.3	0	1,864	
			21	59.9	0	1,610	
M. M.	4 6/12	M	3/15	75.6	35	6,500	
			17	31.5	58	166	
			18	23.3	59	490	
				32.1	34	.....	
			19	23.9	64	594	
			20	25.8	52	220	
			22	44.7	47	1,844	
			23	32.1	50	5,750	
				110.9	0	.....	
			24	69.0	28	3,022	
				48.8	27	.....	
			27	73.7	28	150	
			28	66.8	25	52	
			30	33.4	28	.....	
			4/1	29.6	48	.....	
			3	25.2	49	.....	
			13	34.0	69	.....	

TABLE 2.—*Cases of Meningococcic Meningitis, with Cure—Continued*

Patient	Age	Sex	Date	Spinal Fluid: Lactic Acid, Mg. per 100 Cc.	Spinal Fluid: Sugar, Mg. per 100 Cc.	Spinal Fluid: Total Leukocytes per Cc.	Comment
M. C.	14	M	7/11	78.7	64	13,800	
			12	119.3	26	25,500	
			13	162.4	0	4,800	
			16	63.2	53	155	
				60.1	52	.....	
			21	116.9	14	4,120	
			22	107.1	0	4,210	
			23	98.0	0	430	
				50.4	7	.....	
			25	....	0	260	
			26	76.0	0	80	
			30	66.2	32	213	
			31	52.3	28	30	
			8/ 2	69.2	48	53	
M. C.	2 4/12	M	4/10	86.9	0	2,450	
			11	95.8	0	3,644	
				78.1	7	.....	
			12	76.2	15	8,660	
			14	60.5	25	1,377	
				105.2	0	.....	
			15	85.0	13	780	4/19: blood lactic acid, 42.2; sugar, 122 mg.
			16	51.7	36	1,055	
			17	56.1	52	811	
			18	44.1	43	112	
			19	46.0	19	55	
			28	35.3	32	24	
R. V.	2	M	9/14	78.1	38	5,000	
			16	52.3	Trace	5,500	
			17	46.6	Trace	680	
			18	36.5	17	1,180	
D. F.	7 6/12	M	8/ 6	95.8	5	.....	
				138.6	0	.....	
			7	40.3	92	.....	
			8	34.7	7	1,350	
			9	59.5	45	260	
			13	9.7	69	290	
P. McG.	7	M	7/11	92.2	14	6,700	
G. C.	3 7/12	M	4/ 6	74.3	13	6,022	
				119.7	0	.....	
			7	107.1	0	14,910	
				66.8	27	.....	
			8	126.0	0	7,713	
			10	87.6	39	9,790	
			11	55.4	33	9,000	
			12	32.1	50	258	
			13	36.5	58	1,022	
			14	40.5	72	.....	
			15	35.9	60	.....	
			16	29.6	58	477	
			17	31.8	64	79	
			18	33.7	45	56	
			20	30.2	47	113	
			Blood	22.7	84	.....	
S. B.	1 6/12	..	1/ 3	52.3	15	5,420	
			7	27.7	35	140	

TABLE 3.—*Tuberculous Meningitis*

Patient	Age	Sex	Date	Spinal Fluid: Lactic Acid, Mg. per 100 Cc.	Spinal Fluid: Sugar, Mg. per 100 Cc.	Spinal Fluid: Proteins, Mg. per 100 Cc.	Spinal Fluid: Total Leukocytes per Cc.	Date of Death
M. A.	3	F	4/ 5	61.7	15	.....	50	4/15
D. M.	1 10/12	M	4/ 1	....	..	119.0	400	4/ 9
			4/ 2	....	..	100.0	420	
			4/ 5	69.3	Trace	.....	175	
D. K.	3 10/12	F	5/11	39.1	0	.....	175	5/23
J. F.	1 2/12	M	1/21	67.1	0	86.3	6	1/25
			1/23	78.1	0	....	35	
J. L.	2 11/12	M	6/25	72.5	0	....	68	6/29
			6/27	78.5	0	....	102	
			6/28	81.9	0	....	...	
			6/29	124.1	39	....	205	

TABLE 4.—*Miscellaneous Types of Meningitis*

Patient	Age	Sex	Date	Spinal Fluid: Lactic Acid, Mg. per 100 Cc.	Spinal Fluid: Sugar, Mg. per 100 Cc.	Spinal Fluid: Total Leukocytes per Cc.	Outcome	Comment
J. F.	8/12	M	9/28	150.2	0	500	Died, 9/29	Pneumococcus
R. E.	5/12	M	4/11	107.1	0	1,511	Died, 4/12	B. influenzae
			4/12	105.2	0	3,866		
E. T.	1 9/12	F	1/20	78.7	0	960	Died, 2/ 2	B. influenzae
			1/26	78.1	0	1,110		
A. P.	2 1/12	M	1/18	47.3	34	38	Died, 7/28	B. influenzae and Strep. viridans
			1/20	61.7	0	16		Spinal fluid protein, 42 mg.
			1/28	158.1	0	12,800		Spinal fluid protein, 290 mg.
J. L.	9	M	3/11	101.4	13	1,600	Died, 3/19	Strep. haemolyticus
				143.0	0			
			3/13	144.9	0	9,000		
			3/15	132.9	0			
			3/16	127.9	0	1,780		
D. de Y.	4	M	2/20	27.1	37	870	Unimproved	Strep. haemolyticus
			2/21	27.1	..	160		
			2/23	30.2	27	1,000		
			2/24	....	34	.....	.....	Spinal fluid protein, 71.4 mg.
H. F.	5 10/12	M	6/20	33.4	48	.....	Died, 7/10	Strep. haemolyticus
			6/21	27.7	48	405		
			6/22	59.2	28	4,640		
			6/23	38.4	36	2,400		
			6/24	41.0	21	775		
			6/25	34.7	39	8,200		
			6/26	29.6	48	333		
			6/27	37.8	45	111		
			6/28	30.2	45	.....		
			6/29	29.6	39	220		
			6/30	81.9	0	1,420		
			7/ 1	58.0	0	.....		
H. H.	7	M	10/3	119.0	0	7,500	Died, 10/3	Mastoid; septic meningitis

response of the patient to therapy. Analysis on a quantitative basis was made reliable by the use of Brehme and Brahdy's [5] modification of Clausen's method [6] for the determination of lactic acid. In our experience, this method permits a much greater accuracy than those previously in use, and gives a higher normal range, —16 to 22 mg. per hundred cubic centimeter of whole blood and about 75 per cent of this level for spinal fluid.

During the course of the investigation daily analyses of spinal fluid were made on a group of forty-four patients with meningitis throughout their stay in the hospital. Data on cases are presented in tables 1, 2, 3, and 4. Chart 1 presents graphically the daily variations in sugar and lactic acid of the spinal fluid in a typical case of meningococcic meningitis showing no response to serum therapy and terminating

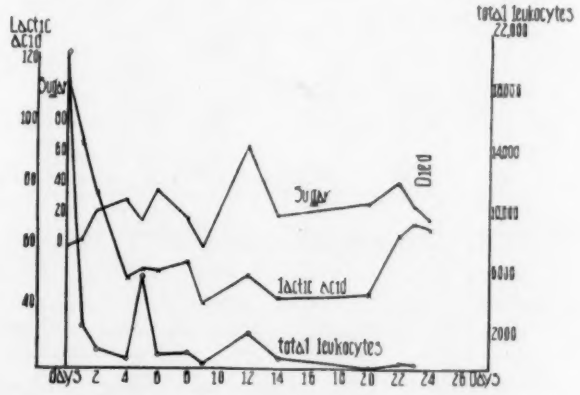


Chart 1.—Observations on the spinal fluid of R. S. The condition was meningococcic meningitis, with death.

in death. The sugar content remained normal, except for two slight increases; the lactic acid, however, remained at a high level. For the forty-eight hours preceding death, there was observed a steady increase in lactic acid. The persistence of the lactic acid at this high level indicated, in our opinion, a failure of response to treatment and poor prognosis. Chart 2 presents the graph for a case of meningococcic meningitis showing an improvement when serum therapy was given. Within a period of twenty-four hours, the value for lactic acid fell from 75 to 30 mg. per hundred cubic centimeters, and throughout the entire course of the observations the curves for sugar and lactic acid showed a reciprocal relationship. With a flare-up of the infection there was observed a precipitate rise in lactic acid and a fall in sugar.

5. Brehme, T., and Brahdy, B.: Biochem. J. **175**:348, 1926.
6. Clausen, S. W.: J. Biochem. **52**:263, 1922.

McQuarrie and Shohl [7] have made a study of the p_H of spinal fluid in relation to the p_H of the blood plasma. They have observed, as have many others, that within the limits of experimental error the p_H of the spinal fluid always approximates that of the blood, except in meningitis. In meningitis the spinal fluid is more acid than the blood. Linder and Carmichael [8] have reported that in cases of cerebrospinal meningitis there is a greater depletion of the base of the spinal fluid than of the blood serum. The explanation of these observations may be found in

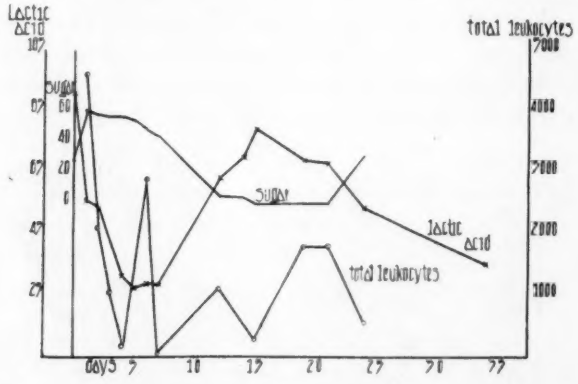


Chart 2.—M. S. had meningococcic meningitis. Recovery occurred.

TABLE 5.—*Comparison of Sugar and Lactic Acid of the Blood and Spinal Fluid in Meningococcic Meningitis*

Patient	Lactic Acid		Sugar	
	Blood, Mg. per 100 Cc.	Spinal Fluid, Mg. per 100 Cc.	Blood, Mg. per 100 Cc.	Spinal Fluid, Mg. per 100 Cc.
V. D.	45.4	146.8	...	0
E. McT.	29.9	128.0	106	0
F. di G.	22.7	97.7	111	0
R. S.	59.9	52.3	74	28
M. C.	42.2	46.0	122	19
G. C.	22.7	30.2	84	47

the greater concentration of lactic acid in the spinal fluid than in the blood in meningitis.

In a variety of pathologic conditions there is observed a rise in the lactic acid of the blood and of the spinal fluid: in some cases of epilepsy, encephalitis, chorea and nephritic convulsions and in acute infections, such as pneumonia and mastoiditis. But in all these conditions the lactic acid of the spinal fluid is less than that of the blood, and varies with that of the blood. In meningitis, however, the lactic acid of the spinal fluid exceeds that of the blood (table 5), and we believe that the increase

7. McQuarrie, I., and Shohl, A. T.: J. Biol. Chem. **66**:367, 1925.
8. Linder, G. C., and Carmichael, E. A.: Biochem. J. **22**:46, 1928.

in the latter is dependent on the increase observed in the spinal fluid. With clinical improvement, as illustrated in chart 2, the lactic acid of the spinal fluid approaches that of the blood. For the determination of pathologic states of the central nervous system, such as meningitis and abscess or tumor of the brain, a knowledge of the ratio

$$\frac{\text{Spinal fluid lactic acid}}{\text{blood lactic acid}}$$

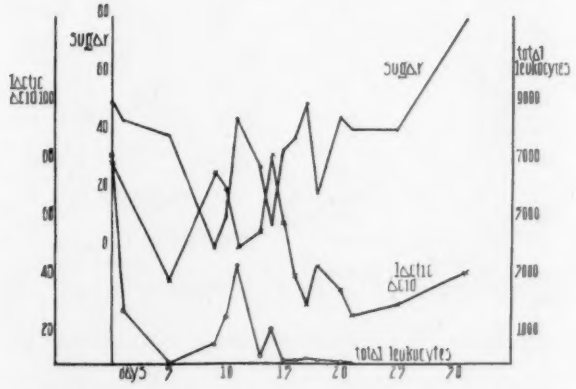


Chart 3.—Curves showing values in a case (R. V.) of meningococcic meningitis in which recovery occurred.

TABLE 6.—*Glycolysis in Spinal Fluid*

Name	Time	Sugar	Lactic Acid	Diagnosis
G. S.	Fresh	72	26.5	Hypertension, no cells, 25 C.
	24 hrs.	73	25.2	
	48 hrs.	76	25.8	
S. R.	Fresh	61	23.7	Hypertension, 38 C., no cells
	4 hrs.	58	25.1	
	19 hrs.	54	22.3	
	47 hrs.	57	27.7	
L. A.	Fresh	103	75.0	Meningococcic meningitis; leukocytes,
	62 hrs.	Negative	144.2	2,216 per cubic millimeter, 25 C.

is a practical diagnostic aid whenever this ratio exceeds 1. The value of the ratio

$$\frac{\text{Spinal fluid sugar}}{\text{blood sugar}}$$

has been discussed frequently in the literature, and data such as these lend further emphasis to the statement that determinations of the sugar of the spinal fluid alone are of little diagnostic value.

The fact that the lactic acid varies directly with the cell count supports the theory that the increased lactic acid of the spinal fluid in meningitis is a product of cellular metabolism. Further confirmation

is afforded by our in vitro experiments on glycolysis in spinal fluid. In table 6 are given the changes in the sugar and lactic acid of two specimens of fluid from persons who were apparently normal in this respect. One specimen was kept at room temperature, the other at 38 C. (101 F.) under aseptic conditions. There was no change in the sugar or lactic

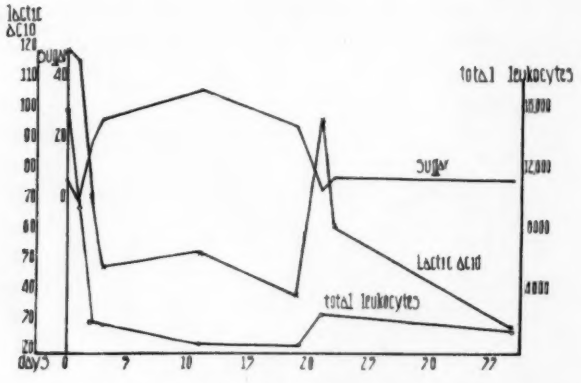


Chart 4.—Curves for H. D., a patient having meningococcic meningitis, with recovery.

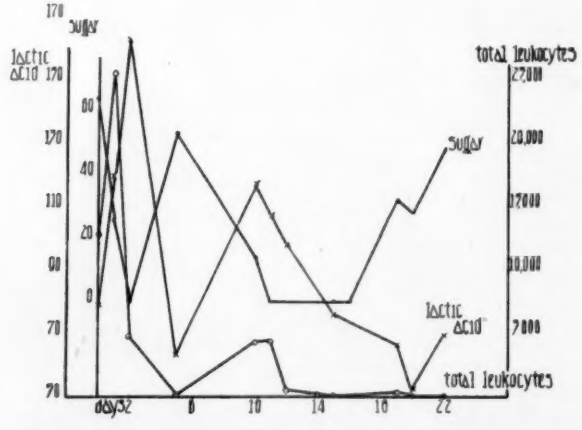


Chart 5.—The patient, M. C., had meningococcic meningitis and recovered.

acid during forty-eight hours. The other specimen of fluid from a case of meningococcic meningitis, with a cell count of 22.16 per cubic millimeter and a high sugar content due to a temporary hyperglycemia, showed during the sixty-two hours of observation, a decrease in sugar and a corresponding rise in lactic acid. The rise in lactic acid accounts for only 70 per cent of the sugar lost. These observations are analogous to the findings in in vitro glycolysis produced by leukocytes reported

by Levene and Meyer [9] or by erythrocytes [10] or spermatozoa.[11] All these observations lend support to our original belief that the rise in lactic acid of the spinal fluid in meningitis is the product of the metabolism of leukocytes.

CONCLUSIONS

Lactic acid is a product of the inflammatory process, and in meningitis the lactic acid of the spinal fluid exceeds that of the blood, and is independent of it. We believe that variations in lactic acid of spinal fluid offer more dependable information concerning the progress of a case of meningitis than any other laboratory procedure at our disposal. Its practical use is further enhanced by the fact that within the last few years the technic of its determination has been greatly simplified and its accuracy increased.

---

9. Levene, P. A., and Meyer, G. M.: J. Biochem. **11**:361, 1912.

10. Garcia, T., and Katayama, I.: Proc. Soc. Exper. Biol. & Med. **26**:122, 1928.

11. Killian, J. A.; McCarthy, J. F.; Stepita, C. T., and Johnston, M. B.: J. Urol. **19**:43, 1928.

CLINICAL EFFECTIVENESS OF A COD LIVER OIL CONCENTRATE

DONALD J. BARNES, M.D.
DETROIT

A concentrate that would retain the virtues of cod liver oil and at the same time permit the giving of a small dose of the substance would be welcomed by infants, parents and physicians alike. Clouse,[1] in an extensive treatise on vitamin D, passed over the use of concentrates with the statement that generally cod liver oil can be given, or, if there is need of a concentrate, viosterol should be used. Physicians in private practice do not feel that concentrates can be treated in such casual fashion. One sees too many infants in whom definite rickets develops in spite of the fact that adequate doses of cod liver oil have been prescribed. Babies do not all take cod liver oil willingly in teaspoonful doses, and some do not tolerate the fat in the quantities used if cod liver oil is given. There is, also, more than a groundless suspicion that these infants do not actually receive the amount of cod liver oil advised.

Data are accumulating which tend to show, further, that viosterol is not the equivalent of cod liver oil, quantitatively or qualitatively. My experience [2] has been that a group of infants receiving 1,250 units of vitamin D in the form of viosterol was no more protected against rickets than an untreated control group, while a group getting approximately the same dose (1,400 units, Oslo) of vitamin D in cod liver oil was protected in 95 per cent of cases. De Sanctis and Craig [3] a little later published a similar study which showed that from 1,400 to 1,700 units of vitamin D given as cod liver oil prevented rickets in 97 per cent of the cases studied, while viosterol 100 D in doses of 3,000 rat units daily prevented rickets in only 77 per cent. De Sanctis and Craig cited their own experience to show that rickets develops in from 25 to 30 per cent of infants not receiving antirachitic agents.

---

1. Clouse, R. C.: Vitamin D, J. A. M. A. **99**:301 (July 23) 1932.

2. Barnes, D. J.; Brady, M. J., and James, E. M.: The Comparative Value of Irradiated Ergosterol and Cod Liver Oil as a Prophylactic Antirachitic Agent When Given in Equivalent Dosage According to Rat Units of Vitamin D, Am. J. Dis. Child. **39**:45 (Jan.) 1930.

3. De Sanctis, A. G., and Craig, J. D.: Comparative Value of Viosterol and Cod Liver Oil as Prophylactic Antirachitic Agents, J. A. M. A. **94**:1285 (April 26) 1930.

Hess, who published favorable results with viosterol therapy, stated in a later paper,[4] "If one has in mind the type of disorder that is generally recognized as rickets, it may be stated that almost complete protection was afforded by this preparation. If, however, one judges by the more refined methods of diagnosis, more especially by a degree of beading of the ribs which, in the course of our study of this disorder, we have learned to associate with rickets, it is found that protection was not absolute." From this experience Hess suggests that the dose of viosterol be doubled or tripled, and this opinion resulted in an increase in potency of the numerous commercial products.[5] That simply increasing the dosage of viosterol did not satisfactorily answer for its failure to prevent rickets was shown by a more recent study by De Sanctis and Craig [6] and one by Hess.[7] There seems to exist a difference other than mere dosage between cod liver oil and viosterol. De Sanctis and Craig [6] fed a total of four hundred infants, divided into five groups, several types of antirachitic agents. One group received 1,500 units (Oslo) daily of cod liver oil; a second group, 3,000 units; a third group, 7,500 units, and a fourth group, 15,000 units of vitamin D as viosterol; a fifth group received 1,500 rat units of vitamin D daily as a concentrate in the form of a tablet. Their results show cod liver oil to be the most effective, the concentrate somewhat less so, while the groups given viosterol indicated that even with the very large dose of vitamin D given as viosterol the protection afforded was not as satisfactory as that obtained with cod liver oil. There is apparently some difference between cod liver oil and viosterol other than the amount of vitamin D measured in rat units contained. Steenbock and his co-workers [8] substantiated this conclusion when they reported that the vitamin D produced by ordinary irradiation of ergosterol with ultraviolet rays from the mercury vapor arc in quartz is a different substance from that found in cod liver oil. Harris [9] said: "The most reliable weapon in

4. Hess, A. F.; Lewis, J. M., and Rivkin, Helen: Newer Aspects of the Therapeutics of Viosterol (Irradiated Ergosterol), J. A. M. A. **94**:1885 (June 14) 1930.

5. American Medical Association, Council on Pharmacy and Chemistry: Increased Potency of Viosterol Preparations, J. A. M. A. **95**:1021 (Oct. 4) 1930.

6. De Sanctis, A. G., and Craig, J. D.: Study of Prophylactic Value of Various Antirachitic Agents, New York State J. Med. **31**:1133 (Sept.) 1931.

7. White House Conference on Child Health and Protection: Growth and Nutrition of the Child, Report of Committee on Growth and Development, New York, Century Company, 1932, p. 157.

8. Steenbock, H.; Kletzien, S. W. F., and Halpin, J. G.: The Reaction of the Chicken to Irradiated Ergosterol and Irradiated Yeast as Contrasted with the Natural Vitamin D of Fish Liver Oils, J. Biol. Chem. **97**:249 (July) 1932.

9. Harris, H. A.: Cod-Liver Oil and the Vitamins in Relation to Bone Growth and Rickets, Am. J. M. Sc. **181**:453 (April) 1931.

the treatment of rickets is cod liver oil." He pointed out that bone formation is a complicated process, and that it is not reasonable that such a process should be governed by a single factor such as vitamin D, which has only the ability to calcify cartilage. Prather and her associates [10] have recently shown cod liver oil to be a more effective antirachitic than viosterol.

The reason for this superiority of cod liver oil as an antirachitic agent is not yet clear, but it is probable that Steenbock [8] is correct in believing that the antirachitic substances in cod liver oil and viosterol are different. It is possible, however, that the presence of vitamin A makes cod liver oil more effective. Apparent recognition of this difference between viosterol and cod liver oil as an antirachitic substance has led to the preparation of certain fish liver oils rich in vitamin A, but relatively poor in vitamin D, to which irradiated ergosterol is added to bring them to the same vitamin D potency as viosterol. So far the efficacy of this method has not been proved. My experience [2] in feeding plain cod liver oil fortified with viosterol would not encourage one to believe that the mere addition of vitamin A, in the levels found in cod liver oil, to viosterol would be enough, since a group of infants so treated were not appreciably superior to a group receiving only plain cod liver oil, though the intake of vitamin D of those on fortified oil was more than two and one-half times that of those on plain cod liver oil.

The general superiority of cod liver oil over viosterol as an antirachitic substance for most purposes seems quite well established. There does remain, however, the perfectly reasonable desire for some satisfactory concentrate. Viosterol, fish liver oils rich in vitamin A with vitamin D supplemented with viosterol and cod liver oil concentrates are all attempts to supply this demand. It has long been known that vitamins A and D could be concentrated in that part of cod liver oil which resists saponification,[11] but only recently have processes satisfactory for the concentration of the vitamins in cod liver oil been commercially feasible. During 1927, the department of agriculture investigated a number of extracts and concentrates of cod liver oil and found that many were almost devoid of vitamin A and only a few contained any material amount of vitamin D,[12] though claims were

10. Prather, E. O., Jr.; Nelson, Martha, and Bliss, A. R.: Viosterol and Cod Liver Oil, Am. J. Dis. Child. **42**:52 (July) 1931.

11. Steenbock, H., and Boutwell, P. W.: The Extractability of the Fat Soluble Vitamin from Carrots, Alfalfa, and Yellow Corn by Fat Solvents, J. Biol. Chem. **42**:131, 1920. Drummond, J. C., and Coward, K. H.: Nutrition and Growth on Diets Devoid of True Fats, Lancet **2**:698 (Oct.) 1921. Drummond, J. C.; Channon, H. J., and Coward, K. H.: Studies on the Chemical Nature of Vitamin A, Biochem. J. **19**:1047, 1925.

12. Cod Liver Oil Stocks Tested for Vitamins (Editorial), J. Am. Pharm. A. **17**:197 (Feb.) 1928.

made that these vitamins were present in abundance. At present, however, concentrates which represent a potency, by animal assays, of several hundred times that of the original cod liver oil can be prepared. Such concentrates are marketed in oil solutions and capsules, as well as in tablets. Marcus [13] has thrown considerable doubt on the stability of the latter, having shown that vitamin A is unstable in the presence of finely divided solids. This may account for the finding of De Sanctis and Craig [6] that the cod liver oil concentrate which they tested was slightly less effective than plain cod liver oil.

Clinical studies on these concentrates, just as has been done with viosterol, must be made if one is to use them in place of or in addition to cod liver oil.

OBSERVATIONS

The concentrate used in the study reported here [14] was prepared in quantity by saponifying a medicinal cod liver oil, dissolving this soap in water and extracting it with a fat solvent. The fat solvent containing the unsaponifiable portion of the oil was distilled to remove the solvent and the residue dissolved in corn oil. This corn oil solution, after preliminary assay, was so diluted that when assayed it showed a content of 20,000 units (Oslo) of vitamin D per cubic centimeter and 50,000 units of vitamin A per cubic centimeter. The vitamin D assay was carried out by feeding young rats the standard Steenbock ration for twenty-one days, followed by a ten day period during which supplemental doses of the cod liver oil concentrate were administered. A definite line of calcification was produced with 0.00005 cc. of the concentrate. The vitamin A content of the concentrate was determined by the usual method of feeding rats deficient in vitamin A supplemental doses of concentrate over a thirty-five day period. The minimal dose producing a gain of 3 Gm. per week was found to be 0.00002 cc.

The prophylactic dosage of antirachitic substance seems fairly well established for cod liver oil at from 1,500 to 2,000 units (Oslo) daily of vitamin D.[15] The size of the curative dose is not so well defined, but it is felt that it must be two or three times the prophylactic dose. Hess [4] stated: "From past experience we believe we should not have seen so many cases of incomplete protection had we given these children 10 teaspoonfuls daily of cod liver oil—in other words, the pre-

13. Marcus, J. K.: The Destructive Action of Finely Divided Solids on Vitamin A, J. Biol. Chem. **90**:507, 1931.

14. The cod liver oil concentrate used in the study was prepared and furnished as an experimental product by Dr. F. W. Heyl, Director of Research Laboratory of Upjohn Company, Kalamazoo, Mich.

15. American Medical Association, Council on Pharmacy and Chemistry: Average Optimum Dosage of Cod Liver Oil, J. A. M. A. **98**:316 (Jan. 23) 1932.

supposed equivalent of 20 drops of viosterol." Ten teaspoonfuls of cod liver oil represents about 5,000 rat units (Oslo) of vitamin D. In another article, Hess [16] referred to the equivalent of 14 teaspoonfuls of cod liver oil, about 7,000 units of vitamin D, as a desirable daily dose in cases of rickets. Other observers recommend dosages expressed in vitamin D units of about from 5,000 to 10,000 rat units daily.[17]

As it was realized that a concentrate that might not be as effective unit for unit as cod liver oil was being dealt with, observations were made on patients with active rickets, and the daily dosage for the three groups studied was 4,000, 8,000 and 12,000 rat units of vitamin D (Oslo), respectively. The oil solution of the concentrate contained 20,000 rat units (Oslo) of vitamin D per cubic centimeter, and was fed from calibrated droppers in daily doses of 0.2, 0.4 and 0.6 cc., respectively.

The patients were obtained from the Detroit Department of Health Child Welfare Clinics and Children's Hospital of Michigan. All the babies studied had active clinical rickets; these findings were supported both by roentgen examination and by determinations of the blood calcium and phosphorus. While the general accuracy of the diagnosis of rickets from the roentgenogram cannot be questioned, it was felt that studies on the blood would give a more accurate picture of early changes, and the roentgenogram definite signs of actual calcification of cartilage. It was originally planned to repeat the blood and roentgen examinations at intervals of one month each, but because of the marked improvement shown even at the lowest dosage level, observations were made at intervals of approximately two weeks. All but nine of the patients had original phosphorus determinations of less than 4 mg. per cubic milliliter. All of these patients had roentgen rickets as well as clinical rickets, and all but two had calcium determinaitons below 10 mg. per cubic milliliter, varying from 9.6 to 6.2 mg.

Quite obviously, the infants who were to be treated needed an adequate diet if they were to recover in a normal fashion from the disease, and consequently each bottle-fed baby was furnished with sufficient milk per day, and each nursing mother with a daily quart of milk. The infants were also given daily adequate amounts of orange juice, so that the results would not be complicated by a deficiency of vitamin C.

16. Hess, A. F., and Lewis, J. M.: Clinical Experience with Irradiated Ergosterol, J. A. M. A. **91**:783 (Sept. 15) 1928.

17. Hess, J. H.; Poncher, H. G.; Dale, M. L., and Klein, R. I.: Viosterol (Irradiated Ergosterol), J. A. M. A. **95**:316 (Aug. 2) 1930. May, E. W.: The Prevention of Rickets in Premature Infants by the Use of Viosterol 100 D, ibid. **96**:1376 (April 25) 1931. Poncher, H. G.: Clinical Use of Viosterol, J. Michigan M. Soc. **30**:258 (April) 1931.

Group 1 (table 1) consisted of thirteen rachitic infants; the table gives the age of the infants in months, the sex, the race and the diet. It gives the weight of the infants in pounds, calcium determinations and phosphorus determinations in milligrams per cubic centimeter of blood, the calcium-phosphorus product, roentgen observations and, under "Comments," the date treatment was started, with other notations pertinent to the progress of the patient. The calcium determinations were made according to the Tweedy modification of the Kramer and Tisdall method, and the phosphorus determinations were made according to the method of Fiske and Subbarow. The infants in this group, in addition to adequate diets, were given cod liver oil concentrate, getting 2 drops three times a day (0.2 cc.), which was equivalent to 4,000 rat units (Oslo) of vitamin D and 10,000 rat units of vitamin A.

Group 2 (table 2) was comprised of fourteen rachitic infants, who were given 4 drops of the cod liver oil concentrate three times a day (0.4 cc.), a dosage equivalent to 8,000 rat units of vitamin D and 20,000 rat units of vitamin A. Table 2 is arranged the same as table 1.

Group 3 (table 3) was made up of fifteen rachitic infants, who were given 6 drops of the cod liver oil concentrate three times a day (0.6 cc.), which was equivalent to 12,000 rat units of vitamin D and 30,000 rat units of vitamin A. The data in this table are arranged the same as in table 1.

Table 4 summarizes the average values for each group, the data being arranged so that the average calcium, the average phosphorus, the average calcium-phosphorus product and the weight given are shown at two week intervals. All patients for whom determinations of the blood were repeated in the first two weeks after treatment was started are reported as having been under treatment for a duration of two weeks. Those who returned between the second and up to the fourth week, inclusive, are considered as under treatment for four weeks.

It is noteworthy that all groups showed a satisfactory gain in weight, and that the average blood calcium and phosphorus returned to normal by the end of the second week of treatment. The average blood phosphorus was identical in all cases at the start of the treatment; the initial blood calcium was highest in group 1; that in group 2 was higher than the average blood calcium in the patients in group 3. All cases, however, had normal average calcium and phosphorus figures at the end of the second week, with a tendency for the calcium to swing to a relatively high level, and then to a slightly lower normal final quantity. The phosphorus in many individual cases showed this same tendency, but not so markedly for the average. It is remarkable that all the infants improved so rapidly, and, while this has not been worked

TABLE 1.—*Effect on Rachitic Infants (Group 1) of Cod Liver Oil Concentrate in a Daily Dosage of 4,000 Rat Units (Oslo) of Vitamin D and 10,000 Rat Units of Vitamin A* *

Number, Name, and Age	Sex and Race	Diet	Date, 1932	Body Weight of Infants	Blood		Calcium Times Phosphorus	Roentgen Observations	Comment
					Calcium	Phosphorus			
2	M	B+C+V	2/23	15.1	12.8	3/6	46.1	R ++	Treatment started 3/3
R. W.	C		3/16	16.1	11.2	4.0	44.8		
6			4/ 2	16.6	11.1	5.5	61.0	Healing	
			4/16	16.3	11.7	6.7	78.4	Healing	
			5/ 2	17.0	10.9	6.7	73.0	Healed	
4	F	F+C+V	2/23	18.6	11.8	3.3	38.9	R ++	Treatment started 3/2
A. J. O.	C		3/23	....	12.5	7.4	92.5		
9			4/ 9	20.3	12.2	6.8	83.0	Healing	
			4/23	20.9	12.0	6.1	73.2	Healed	
			5/ 7	21.3	11.9	7.4	88.1	Healed	
5	M	Full	3/11	24.6	10.8	3.6	38.9	R +	Treatment started 3/3
J. N.	C		4/ 2	24.7	11.0	5.9	64.9	Healed	Cold in head
16			4/16	24.6	11.0	5.0	55.0	Healed	
			4/30	24.7	10.5	4.8	50.4	Healed	
			5/11	25.1	10.8	5.7	61.6	Healed	
8	F	F+C+V	2/23	14.9	12.1	2.3	27.8	R +++	Treatment started 3/3
O. G.	C		3/23	15.8	11.5	6.0	69.0		
7			4/ 6	16.2	11.3	6.0	67.8	Healing	
			4/20	16.8	10.9	4.8	52.3	Healed	Upper respiratory infection
			5/ 6	16.8	11.7	6.1	71.4	Healed	
14	F	F+C+V	2/23	17.1	10.6	3.7	39.2	R +	Treatment started 3/7
C. B. B.	C		3/25	18.2	11.8	7.2	85.0		
10			4/21	19.3	11.7	7.3	85.4	Healed	
			5/ 7	19.5	11.7	6.6	77.2	Healed	
19	M	B+C+V	2/22	17.0	11.6	3.2	37.1	R +	Treatment started 3/3
R. T.	C		4/16	19.8	11.9	6.0	71.4	Healed	Recovered from scarlet fever
7			4/30	20.2	11.5	5.1	58.6	Healed	
			5/14	20.0	10.7	4.8	51.4	Healed	
25	M	B+C+V	2/25	16.9	9.1	3.3	30.0	R +++	Treatment started 3/3
M. S.	C		3/25	16.9	11.1	5.8	64.4		
11			4/ 9	17.2	11.1	6.4	71.0	Healing	
			4/23	17.5	10.9	5.0	54.5	Healing	
			5/ 7	18.6	11.2	5.6	62.7	Healed	
29	M	B+C	3/ 2	13.6	10.7	3.5	37.4	R ++	Treatment started 3/5
R. C.	C		3/30	14.5	11.3	4.6	52.0		
4			4/13	14.3	12.3	5.4	66.4	Healing	Tonsillitis 1 week
			4/27	14.6	11.7	5.5	64.3	Healed	
			5/11	14.8	10.2	4.6	46.9	Healed	
46	M	B+C+V	2/26	19.3	10.3	4.1	42.2	R +	Treatment started 3/2
R. F.	C		3/16	19.8	10.8	3.8	41.0		
8			4/ 5	20.3	12.3	6.6	81.2	Healed	
			4/20	20.7	11.0	6.6	72.6	Healed	
			5/ 4	20.9	11.5	6.2	71.3	Healed	
			5/18	21.0	11.0	6.8	74.8	Healed	
57	M	B+C+V	3/ 3	16.0	9.6	4.1	39.4	R ++	Treatment started 3/7
J. K.	C		4/ 4	17.0	11.5	5.6	64.4	Healing	
5			4/18	17.1	11.5	5.6	64.4	Healing	
			5/ 6	17.4	11.1	6.0	66.6	Healed	
63	M	B+C+V	2/29	16.5	10.1	3.6	36.4	R +++	Treatment started 3/5
G. G.	C		3/29	16.9	10.5	5.2	54.6		
7			4/12	17.1	11.5	5.4	62.1	Healing	
			4/26	17.5	10.9	6.5	70.8	Healed	
			5/10	17.7	10.0	6.0	60.0	Healed	
148	F	B+C+V	3/23	16.1	11.7	3.0	35.1	R +	Treatment started 3/26
B. V.	C		4/ 9	17.0	11.9	5.5	65.4	R +	
5			4/23	17.0	11.6	4.7	54.5	Healing	
			5/ 7	17.5	11.2	5.2	58.2	Healed	
167	F	B+C+V	4/ 4	21.0	10.5	3.0	31.5	R +++	Treatment started 4/6
S. S.	C		4/20	21.1	11.3	4.4	49.7		
9			5/ 4	21.4	12.0	6.7	80.4	Healing	
			5/18	22.0	11.7	6.0	70.2	Healing	

* In this and the succeeding tables, the following are used: Age is expressed in months; sex: M, male; F, female; race: C, colored; W, Caucasian; diet: B, breast-fed; F, formula-fed; V, vegetables; C, cereal; weight is expressed in pounds or a fraction thereof; calcium and phosphorus are expressed in milligrams per cubic centimeter of serum; roentgen observations: R, definite evidence of rickets, the severity being represented by the number of plus signs.

TABLE 2.—*Effect on Rachitic Infants (Group 2) of Cod Liver Oil Concentrate in a Daily Dosage of 8,000 Rat Units (Oslo) of Vitamin D and 20,000 Rat Units of Vitamin A*

Number, Name and Age	Sex and Race	Diet	Date, 1932	Body Weight of Infants	Blood		Calcium Times Phosphorus	Roentgen Observations	Comment
					Calcium	Phosphorus			
16	M	B+C+V	3/16	....	9.2	3.6	33.1	R ++	Treatment started 3/23
V. S.	C		4/ 6	15.3	10.4	4.3	44.7	Healing	
7			4/20	15.1	10.8	5.7	61.6	Healing	Respiratory infection
			5/10	16.0	11.0	5.6	61.6	Healing	
35	F	F+C+V	3/17	18.2	7.8	4.4	34.3	R +	Treatment started 3/21
G. S.	C		4/ 4	19.0	10.7	6.6	70.6	Healing	
5			4/18	19.1	11.4	5.9	67.3	Healing	
			5/ 2	20.0	10.8	4.8	51.8	Healed	
47	F	B+C+V	3/ 7	15.2	11.8	2.2	26.0	R +++	Treatment started 3/12
J. J.	C		3/26	16.1	11.0	4.1	45.1		
7			4/ 9	16.2	12.0	5.4	64.8	Healing	
			4/23	16.8	10.9	4.9	53.4	Healing	
			5/ 7	16.7	12.1	5.0	60.5	Healed	
50	F	B+C+V	3/18	....	10.1	3.5	35.3	R ±	Treatment started 3/23
L. D.	C		4/ 6	16.4	10.7	4.0	42.8	R ±	
7			4/20	17.0	11.2	5.3	59.4	Healed	
			5/ 4	17.6	11.2	5.4	60.5	Healed	
55	F	B+C+V	3/ 5	15.1	10.6	3.2	33.9	R +++	Treatment started 3/11
M. L.	C		4/ 6	16.2	10.2	4.6	46.9	Healing	
7			4/20	16.8	10.8	6.0	64.8	Healed	
			5/ 4	17.4	11.0	5.0	55.0	Healed	
72	M	F+C+V	3/12	....	10.2	4.0	40.8	R +	Treatment started 3/12
C. C.	W		4/28	17.0	11.2	6.2	69.4	Healed	Pertussis
10			5/11	17.2	11.6	6.4	74.2	Healed	
73	M	B+C+V	3/ 7	14.5	9.1	3.2	29.1	R +++	Treatment started 3/12
E. W.	C		4/18	15.7	11.4	5.6	63.8	Healing	
6			5/ 6	15.9	11.6	5.8	67.3	Healed	
79	M	B+C+V	3/ 7	16.4	11.5	2.3	26.4	R +++	Treatment started 3/11
V. J.	C		3/25	17.3	11.0	4.3	47.3		
9			4/11	17.0	11.4	5.7	65.0	Healing	
			4/18	17.3	11.5	5.4	62.1	Healing	
			5/ 2	17.9	10.6	5.8	61.5	Healed	
83	M	B+C+V	3/ 9	16.6	11.3	2.7	30.5	R +++	Treatment started 3/14
B. T.	C		3/28	16.2	11.5	5.2	59.8		
9			4/13	15.4	10.8	5.4	58.3	Healing	Abscess of ear
			4/27	....	....	...	....	Healing	Tuberculin test positive; observations not completed
96	M	B	3/ 9	19.7	7.0	4.6	32.2	R ++	Treatment started 3/16
R. M.	C		3/30	19.2	9.6	5.0	48.0		
9		F+C+V	4/16	20.3	11.5	4.8	55.2	Healing	
			4/30	20.7	10.4	4.6	47.8	Healing	
			5/14	20.6	10.3	4.5	46.3	Healed	
132	F	B+C+V	3/23	10.9	9.5	2.8	26.6	R +++	Treatment started 3/23
R. C.	C		4/ 6	11.7	10.4	4.2	43.7	Healing	
5			4/20	12.2	11.2	4.8	53.8	Healing	
		F+C+V	5/ 5	13.2	12.0	6.2	74.4	Healed	
			5/18	14.0	11.1	7.0	77.7	Healed	
151	M	B+C+V	3/23	18.3	9.2	3.0	27.6	R +++	Treatment started 3/23
M. L.	C		4/ 9	18.7	11.1	4.2	36.6	R ++	
9			4/23	19.0	10.4	5.0	52.0	Healing	
			5/10	19.2	10.6	5.6	59.4	Healing	
152	M	B	3/29	11.2	9.8	3.2	31.3	R ++	Treatment started 3/30
E. A.	C	B+F	4/16	11.4	11.5	4.8	55.2	Healing	
4			4/30	11.6	11.4	5.0	57.0	Healing	
			5/14	12.6	10.7	5.4	57.8	Healed	
153	M	B	3/30	12.7	8.3	4.4	36.5	R +	Treatment started 3/30
W. A.	C		4/16	13.1	11.1	4.6	51.1	Healing	
4		F+C	4/30	13.1	11.1	5.2	57.7	Healed	
			5/14	13.7	10.8	5.4	58.3	Healed	

TABLE 3.—*Effect on Rachitic Infants (Group 3) of Cod Liver Oil Concentrate in a Daily Dosage of 12,000 Rat Units (Oslo) of Vitamin D and 30,000 Rat Units of Vitamin A*

Number, Name and Age	Sex and Race	Diet	Date, 1932	Body Weight of Infants	Blood		Calcium Times Phosphorus	Roentgen Observations	Comment
					Calcium	Phosphorus			
11	F	F+C	3/10	13.8	9.8	3.0	29.4	R +	Treatment started 3/18
C. C.	W		4/ 2	14.1	11.6	6.5	75.4	R +	
8		F+C+V	4/21	14.9	11.5	7.8	89.7	Healing	
			5/10	15.5	11.8	6.5	76.7	Healed	
			5/21	15.5	11.7	6.4	74.9	Healed	
38	F	F+C+V	3/10	10.4	6.2	4.4	27.3	R +	Tetany; Chvostek sign and convulsions; treatment started 3/16
G. H.	C		3/30	11.1	10.5	7.2	75.6		
5			4/13	11.9	10.7	7.5	80.2	Healing	
			4/27	12.9	11.0	7.2	79.2	Healing	
			5/10	13.6	10.7	6.8	72.8	Healed	
76	F	F+C+V	3/ 8	15.2	10.2	3.3	33.7	R +++	Treatment started 3/14
M. F.	C		3/28	16.1	10.4	6.0	62.4		
7			4/11	16.1	11.3	6.4	72.8	Healing	
			4/25	16.7	11.3	6.0	67.8	Healing	
			5/ 9	17.1	11.7	5.4	63.2	Healed	
77	M	F+C+V	3/ 9	22.4	10.1	3.6	36.4	R +++	Treatment started 3/15
W. T.	C		3/24	22.7	10.5	6.6	69.3		
13			4/11	24.2	11.9	6.1	72.6	Healing	
			5/ 2	23.8	10.9	7.2	78.5	Healed	
86	M	F+C+V	3/10	13.2	10.9	3.6	39.2	R ++	Treatment started 3/16
G. V.	W		3/30	14.0	10.2	6.4	65.3		
7			4/16	14.3	11.8	5.5	64.9	Healing	
			4/30	15.4	11.2	6.4	73.7	Healed	
			5/14	16.0	11.8	5.4	63.7	Healed	
89	F	B+C	3/10	15.6	9.9	2.8	27.7	R +	Treatment started 3/16
O. P.	C		4/13	17.1	12.8	6.1	78.1	Healing	
5		B+C+V	4/27	17.7	11.7	5.4	63.2	Healed	
			5/11	18.0	11.8	5.3	62.5	Healed	
91	M	B	3/10	11.6	7.1	4.7	33.4	R +	Treatment started 3/16
C. D.	C		3/30	12.1	11.2	6.6	73.9		
4		F+C	5/ 2	13.4	10.7	5.6	59.9	Healed	Recovered from pertussis
			5/16	14.0	11.2	6.3	70.6	Healed	
102	F	B	3/14	14.9	10.3	3.6	37.1	R +	Treatment started 3/21
J. M.	C	B+C+V	4/ 4	14.1	11.1	6.0	66.6		
6			4/18	14.2	12.0	6.1	73.2	Healed	Recovered from pertussis
			5/ 2	14.4	11.3	5.2	58.8	Healed	
114	M	F+C+V	3/17	13.6	10.1	2.9	29.3	R +++	Treatment started 3/21
T. B.			4/ 4	14.1	12.4	4.4	54.6	Healing	
8			4/20	13.9	10.8	4.9	52.9	Healing	Cold in head; poor appetite
			5/12	14.5	11.2	5.4	60.5	Healed	
121	M	B	3/19	11.2	10.1	3.2	32.3	R ++	Treatment started 3/19
R. R.	C		4/11	11.9	11.6	3.6	41.8	Healing	
4		B+C	4/23	12.2	11.8	4.0	47.2	Healing	
			5/ 7	12.6	12.1	4.8	58.1	Healing	
			5/21	13.2	11.3	5.9	66.7	Healed	
134	M	B+C+V	4/ 1	12.3	9.1	4.4	40.0	R +	Treatment started 4/1
R. S.	W		4/13	12.4	10.2	4.2	42.8	R +	
5			4/27	12.9	11.7	5.6	65.5	Healing	Vomiting
			5/11	13.6	12.1	5.4	65.3	Healed	
136	M	B+C+V	3/22	14.6	10.4	2.0	20.8		Treatment started 3/26
O. D. W.	C		4/ 9	14.8	11.1	6.0	66.6	Healing	
9			4/23	15.1	11.9	5.4	64.3	Healing	Vincent's angina
			5/ 4	15.1	11.3	5.6	63.3	Healed	
143	M	B+C	3/29	15.2	9.3	3.3	30.7	R +++	Treatment started 3/31
C. G.			4/11	15.9	10.7	4.3	46.0	Healing	
7			4/26	16.4	11.2	6.0	67.2	Healing	
			5/18	16.9	11.2	5.7	63.8	Healed	
154	F	B+C+V	3/31	15.1	11.1	2.4	26.6	R +++	Treatment started 3/31
S. W.	C		4/11	15.6	10.9	3.8	41.4	R +++	
10		F+C+V	4/23	15.2	10.7	4.4	47.1	Healing	
			5/ 7	15.7	11.4	5.2	59.3	Healing	
			5/21	15.9	11.5	5.1	58.6	Healed	
160	M	F+C+V	4/ 2	17.5	9.4	3.6	33.8	R ++++	Treatment started 4/2
C. W.	C		4/16	18.6	10.5	6.5	68.2	R ++++	
16			5/ 6	19.1	11.0	6.1	67.1	Healing	Rickets of spine
			5/21	19.9	10.7	5.8	62.1	Healing	

out in table form for the roentgenograms, in several cases undoubted healing, as evidenced by calcifying cartilage, had begun by the second week of treatment. The following cases are examples: O. D. W. (table 3, case 136) started treatment on March 26, 1932, and showed healing on April 9. M. L. (table 2, case 151), started treatment on March 23, 1932, and showed healing on April 9. V. S. (table 2, case 16), showed healing after two weeks of treatment, as did G. S. (table 2, case 35).

From table 4, charts 1, 2 and 3 were prepared, showing the curves for the calcium, phosphorus, weight and calcium-phosphorus product. The initial average determinations for blood calcium and phosphorus

TABLE 4.—*Summary of Average Values for Each Dosage of Vitamin D*

Vitamin D, Units (Oslo) Daily	Duration of Treatment, Weeks	Blood		Calcium Times Phosphorus	Body Weight of Infants, Pounds
		Calcium, Mg. per Cc. of Serum	Phosphorus, Mg. per Cc. of Serum		
4,000	0	10.9	3.4	37.0	18.5
	2	11.6	5.5	63.9	19.2
	4	11.4	5.7	65.0	19.5
	6	11.5	6.0	69.0	19.7
	8	11.2	5.8	65.0	20.1
	10	10.9	5.9	64.3	20.2
8,000	0	9.7	3.4	33.0	15.3
	2	10.8	4.7	50.8	15.9
	4	11.1	5.2	57.7	16.2
	6	11.0	5.5	60.5	16.8
	8	11.0	5.6	61.5	17.1
12,000	0	9.5	3.4	32.3	14.4
	2	10.9	5.7	62.1	15.1
	4	11.5	5.8	66.6	15.5
	6	11.3	5.8	65.5	16.1
	8	10.4	5.8	60.4	16.5

and those made at two week intervals after treatment was started are shown for each group. Charts 1 and 3 show a tendency for the calcium to rise to a high level and then recede to an average normal. Chart 2 shows a tendency in this respect, but the calcium does not drop as low as it does in charts 1 and 3. The phosphorus does not exhibit a similar decline, but reaches a level, where it remains. The weight throughout shows a progressive increase. No deficiencies were noted in the study according to the type of feeding. The groups were quite comparable as to age, and the patients were all followed up closely, being visited at least once a week in the home by the nurse detailed to this study, and being seen about every two weeks in the clinic. There were no difficulties in administering the medication, and the parents of the patients cooperated well, showing unsolicited enthusiasm at the rapid progress of the patients, which was manifested chiefly in the increased activity and gain in strength.

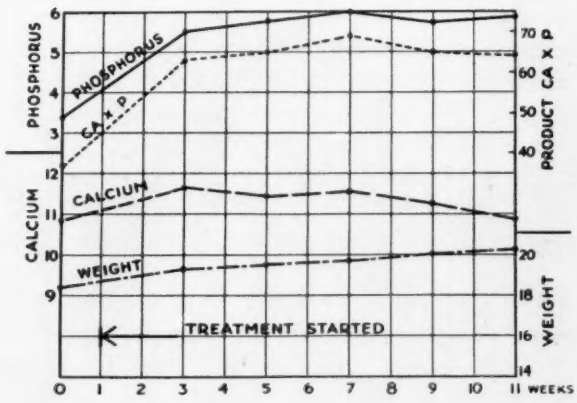


Chart 1.—Curves with daily dosage of 4,000 rat units of vitamin D.

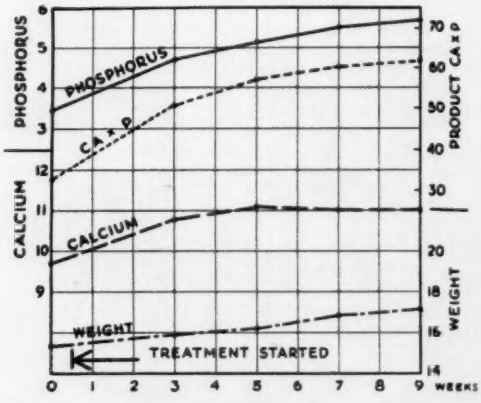


Chart 2.—Curves with daily dosage of 8,000 rat units of vitamin D.

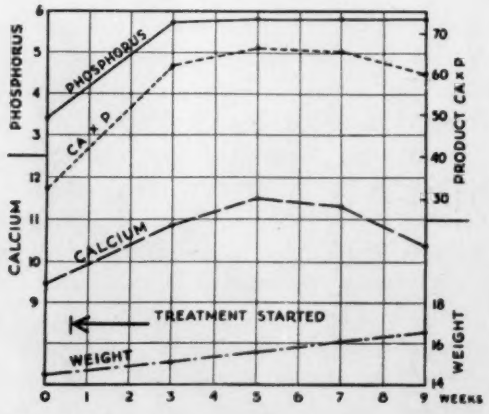


Chart 3.—Curves with daily dosage of 12,000 rat units of vitamin D.

CONCLUSIONS

1. Forty-two rachitic infants were divided into three groups of thirteen, fourteen and fifteen, respectively, and they were given each day 6, 12 and 18 drops, respectively, of a corn oil solution of cod liver oil concentrate of such a potency that they received, according to groups, 4,000 units of vitamin D and 10,000 units of vitamin A; 8,000 units of vitamin D and 20,000 units of vitamin A; and 12,000 units of vitamin D and 30,000 units of vitamin A.

2. Healing was very rapid in all cases and was borne out by the return of the blood calcium and phosphorus to normal within an average period of two weeks or less and a rapid increase in strength and activity on the part of the child. Roentgenograms showed rapid calcification, this roentgen evidence paralleling the improvement in the blood picture.

3. The medication was well taken and caused no dietary disturbances.

4. The healing was due to the cod liver oil concentrate, and not due to a loss of weight or a stationary weight, as is shown by the progressive gain all children showed. That it was not due to the effect of the sun's rays was evident by the fact that patients with active rickets who showed no healing were continuously coming to the clinic up to the time the experiment was concluded.

5. This cod liver oil concentrate retains its active antirachitic effectiveness as measured in rat units of vitamin D, and, in the dosage levels studied, is remarkably effective in curing rickets in infants.

POLIOMYELITIS

COMPARISON BETWEEN THE EPIDEMIC PEAK AND THE HARVEST PEAK

JOHN A. TOOMEY, M.D.
AND
MYRON H. AUGUST, M.D.
CLEVELAND

Elsewhere [1] we plotted the incidence curve of infantile paralysis for Cleveland and vicinity, a curve which had its peak in the months of August, September and October. Aycock and Eaton [2] have called attention to the biseasonal prevalence of the disease in the United States, and their figures show a very evident spring hump in the morbidity rate. Such a spring incidence might pave the way for a subsequent seasonal epidemic. We have not experienced a noticeable increase of cases in our community in the spring. It is probable, however, that if many districts such as ours were lumped together this increase of cases in the spring would become more obvious.

Our local hospital and the board of health reports are slightly at variance with each other, the former with few reported cases in the winter and the latter with a greater number. Many patients are admitted to the hospital in the winter months with various afflictions incorrectly diagnosed as poliomyelitis. Accuracy of diagnosis is not always obtained in the stress of general practice, and many unrelated neurologic conditions may be included under the term infantile paralysis. One does not infer from this that there are no cases in the winter, but one is entitled, at least in our immediate locality, to question the correctness of this diagnosis when it is made between January and June—this in spite of the fact that Aycock and Eaton reported a surprising infectivity throughout all the months of the year.

The seasonal epidemic periodically of the disease seems uniform throughout the United States and Canada, save in California, where the incidence peak is prolonged over a number of months.

Temperature has no effect on the morbidity of the disease, since the maximum peak of the former came on earlier in the year than did the maximum peak of the latter.[1]

From the Department of Pediatrics, Western Reserve University, and the Division of Contagious Diseases, City Hospital.

1. Toomey, J. A., and Lipson, H. A.: The Seasonal Incidence of Poliomyelitis in and About Cleveland, J. Pediat. **1**:739 (Dec.) 1932.

2. Aycock, W. L., and Eaton, Paul: The Biseasonal Prevalence of Infantile Paralysis, Am. J. Hyg. **4**:356, 1924.

The majority of epidemics are reported above and below 30 degrees latitude north or south. Location seems to affect the incidence of the disease, for it is not usually present in epidemic proportions in the torrid zone. Though the disease is peculiarly a disease of the temperate zone, there are many places above 30 degrees latitude where it is rarely epidemic. This is easily understood when one remembers that the heat or coolness of a locality depends on many factors other than distance from the equator. For this reason, local topographical conditions may modify the climate to such an extent as to make a region temperate by position and torrid in actuality.

We have shown that the disease could not be affected by either the maximum or the minimum amount of sunlight present in our locality [1] since the disease occurs with extreme regularity some time after the summer solstice in localities in the northern hemisphere. Smith [3] has shown that the height of the curve of the angle of the sun (about latitude 37 degrees north) in the northern hemisphere is in June, a time prior to the usual incidence of the disease.

Climate seems to be a factor. Florida and the southeastern states have had few cases, while California has had many. The latter state, which has a mountain barrier situated a little inland from the sea and an intervening area swept by the westerly trade winds, has a fairly equable climate. Florida, a little lower in latitude, is more torrid by comparison during the usual poliomyelitic season.

Accurate statistics are not available for most of the Asiatic countries, but one would hardly expect that epidemics could be unnoted in this day and age even if they happened to occur in remote districts. Over half of China is above 30 degrees latitude, and terrific heat is experienced during the summer and intense cold in the winter. Here one would expect a marked increase in cases, but the number of those reported is minimal. One case was found in 25,000 admissions to Peiping Union Medical College Hospital.[4]

Due west of China is Japan, a land that has one hundred and fifty days of either rain or snow and two hundred and fifteen days of sunshine. It is not so cold as the mainland, since the Siberian winds are warmed over the intervening sea, and precipitation is thus massive along the eastern slope. Up to 1926, the disease was not even mentioned in the Tokyo yearbook, although the League of Nations' report [5] called

3. Smith, James H.: The Influence of Solar Rays on Metabolism, Arch. Int. Med. **48**:907 (Nov.) 1931.

4. Zia, S. H.: The Occurrence of Acute Poliomyelitis in North China, National M. J. China **16**:135, 1930.

5. League of Nations Epidemic Report on Poliomyelitis, Monthly Epidemiol. Rep. **11**:1, 1932.

attention to some cases in 1929, when 117 deaths were reported. One wonders how the morbidity is so low in a state that teems with a population of 84,000,000.

Theoretically, the disease should occur at about the same latitude south of the equator, and the height of the incidence curve of the disease should be about the same number of days distant from the vernal equinox in the southern hemisphere as ours is from our equinox.

In examining the south temperate zone, we find that below 30 degrees latitude, there are the following habitable lands: Australia, New Zealand, the Argentine Republic, Chile, Uruguay and the tip of Africa, i. e., parts of the Union of South Africa.

Though mortality rates were available, morbidity statistics were not obtained from South American countries. During the past ten years only 9 deaths occurred in Rio de Janeiro, a city of 1,729,000 people. The Brazilian climate ranges from tropical and semitropical in the north to temperate in the three lower states, districts in which there may even be snow. The fact that a state with a population of over 30,000,000 (census of 1930) had so few deaths is interesting, and it is theoretically what one could expect from the location if latitude constitutes a direct or indirect factor.

Uruguay (population, 1,042,668) has had small epidemics. This is not surprising when one remembers that a mild temperate climate is found in this state, extending as it does from 30 to 35 degrees latitude south in juxtaposition to Brazil, along the southern border of the latter.

In Chile (population, 3,947,374) there is a desert north of 30 degrees, and the land experiences a rainy season south of 40 degrees. The contour of the country is unlike that of other South American states, as the only really vegetative district is the elevated central zone, the vale of Chile, hemmed in by the Andes on one side and the coastal range on the other. Heat in the north, rain in the south and high altitude in the center might have some effect on poliomyelitis indirectly, by causing changes in the temperature curve. The number of reported cases is not great.

Theoretically, the Argentine Republic should have the greatest morbidity rate in South America, with few cases in the northern marsh region and in the cold, comparatively uninhabited steppes below the Rio Negro. From the mortality rates furnished us by Vaccarey and Luccini [6] of the National Department of Hygiene at Buenos Ayres, we may infer the occurrence of epidemics.

6. Vaccarey and Luccini: Personal communication to the authors.

The southeastern part of Australia is below 30 degrees latitude and is the coolest portion of the island, owing to the presence of the highlands. Ground frost may even occur, but snowfall is normally limited to the southeast headlands and to Tasmania. The climate is typically temperate along the southeastern coast from 34 to about 44 degrees latitude south (including Tasmania). In western and central Australia the climate is decidedly different, and the heat terrific. One would expect to find some cases in the southern part of Queensland, in New South Wales, Victoria and Tasmania, while few should be found in western and central Australia.

The whole of New Zealand is below 34 degrees latitude, but the northern tip of the island is semitropical, though in the region of 37 degrees latitude. This should be and is a point of occasional infection.

Theoretically, there is no other land in the southern hemisphere below 30 degrees latitude where poliomyelitis should occur, save at the very tip of Africa. We find, however, definite reports of poliomyelitis from the Transvaal, 25 degrees from the equator. When we examine the physical contour of the eastern tip of Africa, we find the land from 4,000 to 5,000 feet high, an inland plain. Because of this South African plateau, 40 per cent of the land in the tip of Africa east and south of the Kalahari desert is over 4,000 feet above the sea level, giving the area a climate that is cooler by far than that of many states situated farther from the equator in the north temperate zone. When one figures a drop of 1 degree in temperature for every 150 feet in elevation, the variation in temperature immediately becomes understandable.

Curious climatic variations are seen, especially in Natal, where the narrow coastal strip is subtropical, while less than 100 miles inland from Durban the land rises sharply and the climate becomes totally different, passing from a subtropical to a temperate area within these few miles. Such places could have poliomyelitis.

When we analyze the curves of incidence from Italy, Hungary, Austria, Latvia and Switzerland (chart 1), which are above the equator, and those from New Zealand, Australia, etc., in the southern hemisphere (chart 2),[5] we note that most epidemics occur with fairly even regularity in either hemisphere at a certain time after the vernal equinox.

Epidemics seem to occur a little sooner the nearer the locality is situated to the equator, so that Aycock [7] has described a "southern" type of incidence peak, an incidence peak which starts a little before the morbidity peaks in the more northern parts of the United States. The same author noted the peculiarities of California epidemics and classified them in his southern category.

---

7. Aycock, W. Lloyd: A Study of the Significance of Geographic and Seasonal Variations in the Incidence of Poliomyelitis, Am. J. Hyg. **3**:245, 1929.

Though the climate in a rough way seems to limit the peak of the disease to certain months in the north and south temperate zones, it does not explain the seasonal incidence.

The thing common to all these countries is their vegetation (exceptions will be noted later). It is an old opinion (Wickman [8]), that this disease is of gastro-intestinal origin (Kling [9]). Because of this, one might find some valuable information in a study of the vegetation of these various lands.

Roughly, the northeastern part of the United States, the seaboard states including Virginia, the midplain area down to Texas, the north central states including Kentucky, the northwestern states and the upper portion of California are devoted to practically the same kind of agriculture and fruit husbandry. The greater part of California is planted in "perishables." These states have epidemics of poliomyelitis. South of Tennessee and Virginia and east of the Mississippi, the commercial fruit industry is different save in the peach district of Georgia, and the raising of grains is not a major undertaking. Even though one of the first descriptions of the disease was from a Louisiana parish, epidemics are not so common in the southeastern parts of the United States as they are in the northern.

All European countries, save Italy and Spain, are agriculturally like the northern part of the United States. Italy and Spain have hotter climates. In northern Italy and Spain, north temperate vegetation is grown; in the south, the vegetation of a hotter climate. Curiously in the epidemic of 1929, most of the cases occurred in northern Italy.

China has vegetation like ours, in addition to other indigenous products like the soy bean. Japan, with her intensive production of rice, definitely differs agriculturally from other countries of the temperate zone.

In the other hemisphere the same conditions prevail. Brazil supports north temperate vegetation only in the southernmost tip of the three lower states and, as might be expected, has relatively few cases, although it is more densely populated than other regions of South America.

Uruguay is in reality one of the old southern Brazilian states. The Argentine Republic has crops of wheat, maize, oats, barley and potatoes and all the fruits, such as plums, peaches and pears, and is therefore like the northern part of the United States in husbandry. Chile, although in the proper latitude, because of limitation of soil and other

8. Wickman, Ivar: Acute Poliomyelitis (Heine-Medin's Disease), translated by W. J. M. A. Maloney, New York, Journal of Nervous and Mental Diseases Publishing Company, 1913.

9. Kling, C.: Investigations on the Epidemiology of Poliomyelitis, Bull. office internat. d'hyg. pub. **20**:1779, 1928.

topographical reasons, cannot support as plentiful a vegetative season as her neighbor.

In Australia, the states of New South Wales and Victoria, as well as Tasmania and the islands of New Zealand, have an agricultural productivity like the northern part of the United States.

Because of the height of the South African plain above the sea, the temperature is modified, so that the inhabitants are able to grow maize, Kaffir corn, wheat, oats, barley, apples, peaches, etc., just as in the Argentine, which is much below South Africa in latitude.

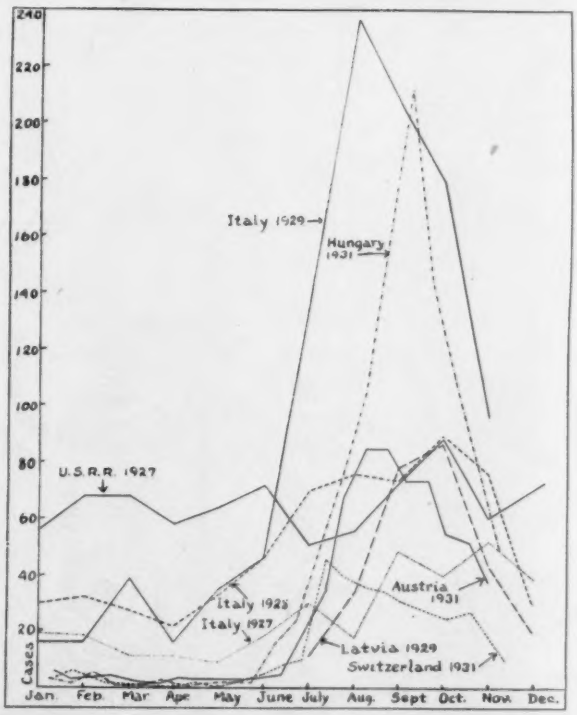


Chart 1.—Seasonal incidence of poliomyelitis in some European countries.

In conclusion, one could say that the disease might occur, but is not prevalent, in the torrid zone. When it occurs epidemically, it occurs at the same time each year (the exceptions are California,[10] Union of Soviet Socialist Republics[11] and Uruguay.[12] The disease usually

10. Porter, G. S., California: Report of 1930 Cases by Month, Sacramento, Calif., Department of Health.

11. League of Nations Epidemic Reports on Poliomyelitis in Union of Socialist Soviet Republics, Monthly Epidemiol. Rep. **9**:47 and 97, 1930.

12. Marquio, L.: Winter Incidence of Heine-Medin's Disease, An. Fac. de med., Montevideo **8**:7, 1923.

recurs only in those countries which raise the same type of agricultural products.

Is there anything to connect growth with this disease? Previously, we [13] thought we saw a lead in this connection, and unsuccessfully tried to determine some factor that would affect vegetation in the months of

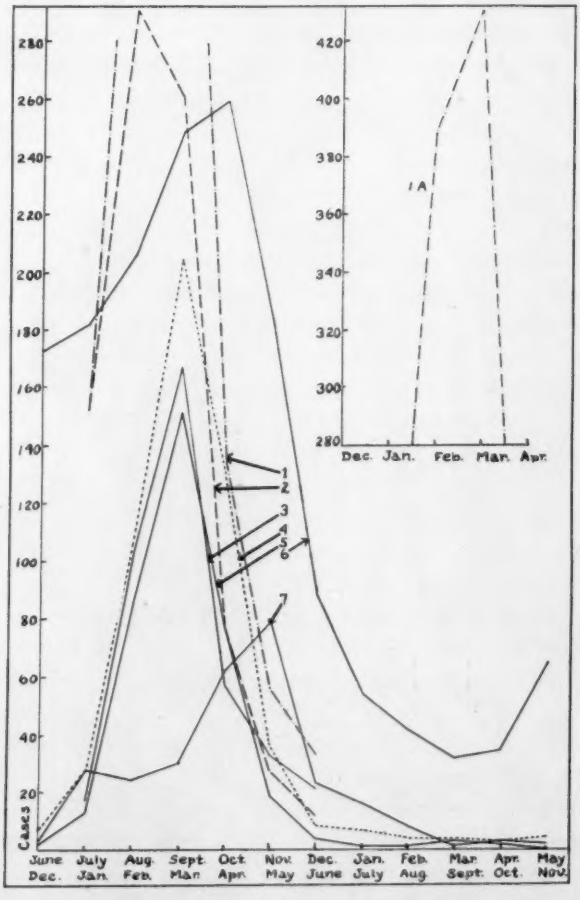


Chart 2.—Incidence curves of poliomyelitis for some north and south temperate areas: *1*, total number of cases in New Zealand, North and South Island, in 1924 and 1925; *1A*, top of curve; *2*, cases in New Zealand, North Island; *3*, cases in New Zealand, South Island; *4*, cases reported by the Cleveland Board of Health from 1921 to 1931; *5*, cases in the Cleveland City Hospital from 1921 to 1931; *6*, cases in New South Wales from 1921 to 1931; *7*, cases in Victoria, Australia, in 1931.

13. Toomey, J. A., and August, M. H.: Temperature and Precipitation in Relation to the Morbidity Rates of Poliomyelitis, J. Pediat. **1**:601 (Nov.) 1932.

growth. One of our colleagues, Dr. C. W. Burhans, thought that green apples were a factor. The regularity with which the incidence peak occurs prompted us to study the time interval between the vernal equinox and the advent of fructification and then to compare the data with the incidence curves for poliomyelitis in the districts studied.

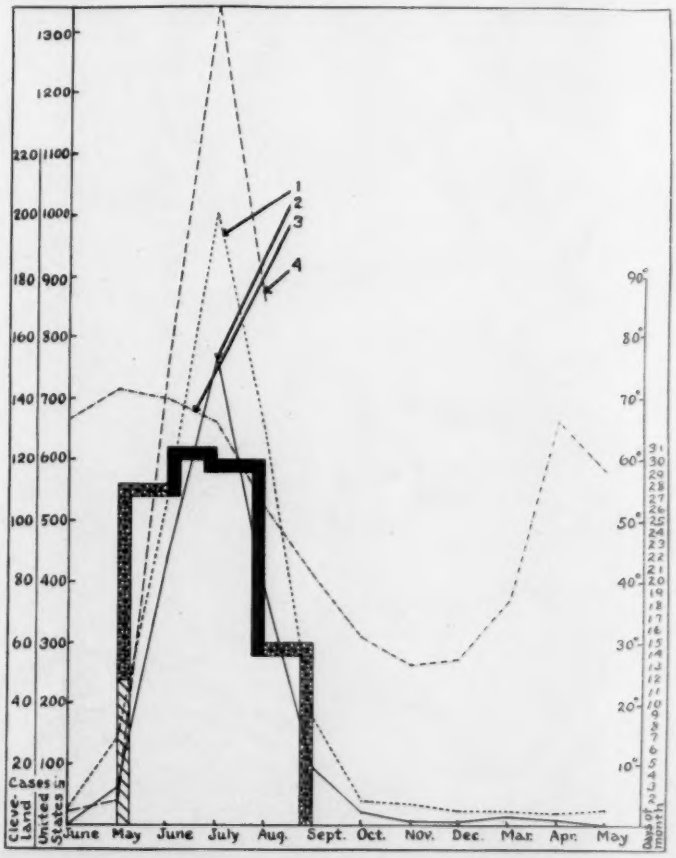


Chart 3.—Seasonal incidence of poliomyelitis in Cleveland from 1921 to 1931, compared with data on harvest: *1*, cases reported by Cleveland Board of Health; *2*, cases in City Hospital; *3*, mean temperature in Cleveland; *4*, cases in the United States in 1931 (report for December is lacking). In this and charts 4 to 7, inclusive, the entire blocked area represents the total vegetative season; the criss-cross blocks indicate the total apple season, and the black blocked area the harvest peak for apples.

For this study, one must consider the growing time of grains, fruits and vegetables in various countries and in different latitudes. The interval between the last spring frost and that coming first in the fall constitutes the growing season anywhere. Such information is avail-

able for the United States, but it was soon found that meteorological services of countries other than our own have a different conception of what constitutes frost. Hence the data from North and South American and European countries on frost are not comparable.

It was thought best, then, to start computations from the maturing time of the earliest general crop. The incubation or actual growing time prior to maturity would not be important in our considerations, since

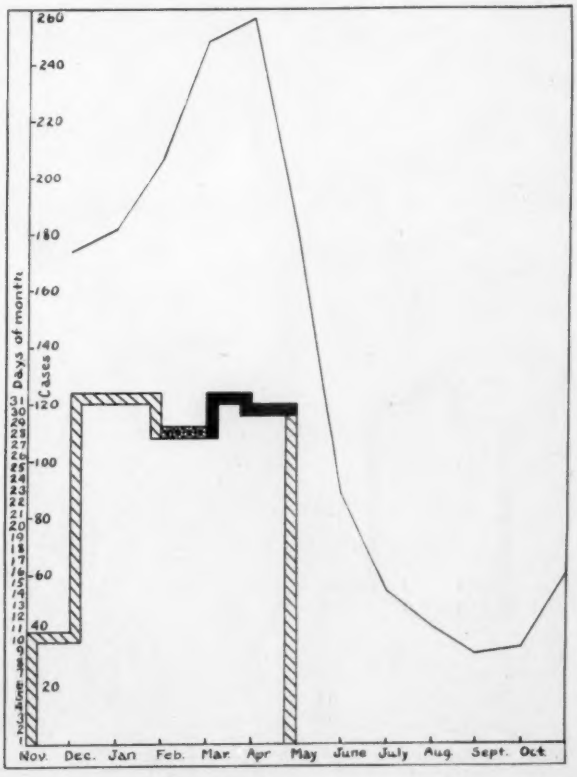


Chart 4.—Seasonal incidence of poliomyelitis in New South Wales from 1913 to 1931, compared with approximate data on harvest.

the maturing dates were the vital factors. Wheat was chosen, since it usually matures first and is planted throughout practically all the zones in which poliomyelitis occurs. In the United States, it is harvested any time from the end of May in the mid-Southwest to July 15 in the Northwest. It was thought that the first harvesting dates of wheat could be taken as the date of the earliest maturing general crop of a district. There is probably vegetation that grows faster in certain sections, such as lettuce, but accurate information about these products is lacking.

For the last maturing data of the season, we should have to pick a product that would be generally found in the temperate zone, a product about which there was some accurate information in obtainable form. The date of the last apple picking could be used. It is true that peaches and pears are sometimes picked later, but accurate information on these crops is not so readily available as are the various dates for apples. In those places where wheat is not grown to any

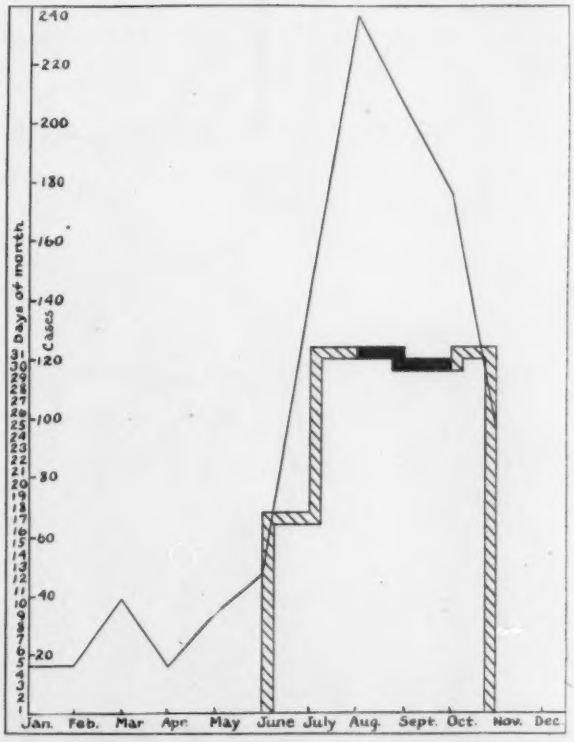


Chart 5.—Seasonal incidence of poliomyelitis in Italy for 1929, compared with approximate data on harvest.

great extent, as in California, the dates of apple bearing could be used as an approximation of the total period of growth for the district.

Though wheat is probably one of the earliest of the crops harvested, it could hardly cause gastro-intestinal reactions, since its availability for consumption is delayed. The same comment could apply to other grains, such as rye. Their maturing dates likewise do not correspond with the incidence dates of the usual epidemics. The harvested crops that might cause intestinal upsets are the perishables, and for our purpose we would have to know their period of growth. A short time after

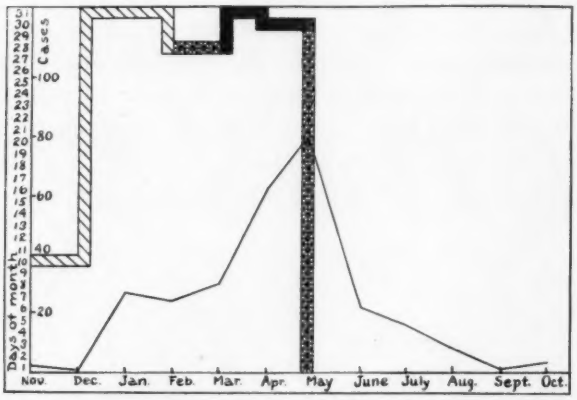


Chart 6.—Seasonal incidence of poliomyelitis in Victoria epidemic for 1931, compared with approximate data on harvest.

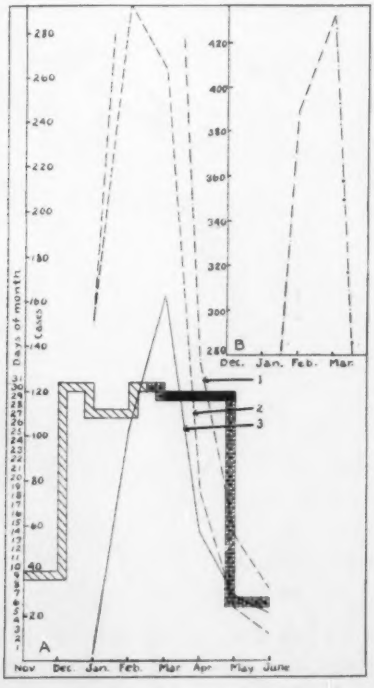


Chart 7.—Seasonal incidence of poliomyelitis in New Zealand in 1924 and 1925, compared with approximate data on harvest. *A, 1,* total number of cases in New Zealand, North and South Island; *2,* cases on North Island; *3,* cases on South Island. *B,* top of curve *1.*

the wheat crop is harvested in northern Ohio, or about July 16, the first apples are picked, and this crop is continually harvested up to October 15. Within these inclusive dates fall the fructification or ripening dates of every perishable picked, and this crop is continually harvested up to October 15. Our first thought was that if the date for the harvesting of wheat was known, the approximate first date of maturing of the apple could be figured out, and the latter could be regarded as the first date for the maturing of the "perishables." This dictum was not

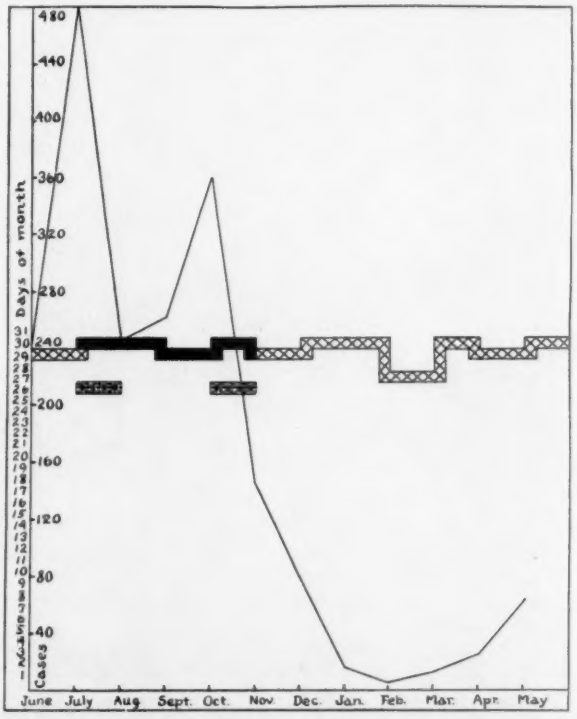


Chart 8.—Seasonal incidence of poliomyelitis in California for 1930, compared with approximate data on harvest. The cross-blocked area indicates the vegetative season (all year) and the black area, the apple harvest season; the criss-cross blocks represent the apple harvest peaks for the northern and southern districts.

agreed to by experts in the Foreign Fruit and Vegetable Department of the United States Bureau of Agriculture (personal communication); hence, with their aid, we constructed a table of the actual earliest dates of maturing for the perishables and, for our purposes, considered the first date of maturing of the apple as the first date of maturing of "perishable products."

A third bit of information, which is most important of all, is the harvesting, picking or peak date of the "perishable vegetation" in a locality. The harvest peak of the apple crop in our locality occurs about the same time as the harvest peak of most other vegetation. This is probably, in a general sense, true of other localities, since the number of days necessary for the growth of the various fruits and vegetables is fairly uniform for the same type of climate, although a wide range in the rapidity of growth is admitted.

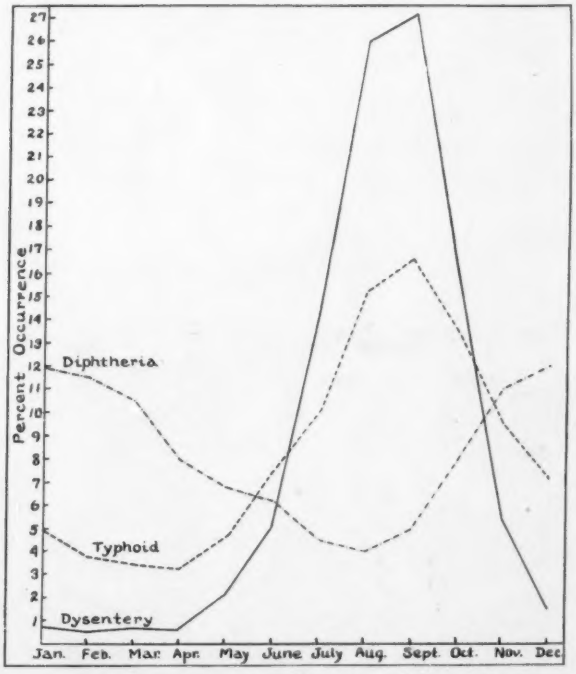


Chart 9.—Diphtheria, typhoid and dysentery curves (from data appearing in the Japanese Sanitary Bureau Annual Report, 1925-1926).

Three sets of data, then, were important: first, an inclusive set which would represent the maturing season of all vegetation; second, an inclusive set of dates which would represent the entire maturing season of apples, and third, an inclusive set of dates which would represent the harvest peak of apples. The first set of dates would represent the total harvest season; the second, the total harvest season of perishable vegetation, and the third, the harvest peak of perishable vegetation.

Definite information as to the actual harvest peak in many countries was lacking, and the information obtained was drawn from consular

reports on the foreign fruit export trade.[14] Bearing this in mind, one can see that there may be slight differences in the peak time of any of the three curves one way or the other, and that our curves are approximations. Especially is this true when we lack specific information on the physical topography of the land in question and have to rely on general information.

The data obtained were plotted against some epidemic poliomyelitis curves of our community and of other communities. It must be borne in mind that the epidemic and not the endemic curves were studied.

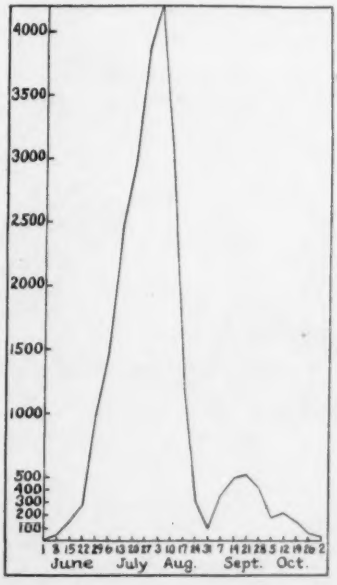


Chart 10.—Seasonal increase of "flies," (from data given by Johnson, G. A.: J. Am. Water Works A., 1916, vol. 3, nos. 2 and 3).

When we take the harvest peak of the apple curve in our locality and plot it against the incidence curve of cases, we find that the height of the harvest curve occurs at the same time as the peak of the incidence curve of infantile paralysis (chart 3).

When we plot the various epidemics in other lands in the same way (charts 4, 5, 6 and 7) we again see the near coincidence of the incidence curves of poliomyelitis with the harvest curves. The Union of Soviet Socialist Republics, in the north temperate zone, seems to

14. The dates of the maturing of wheat and apples for the state of California and for countries other than our own were obtained from the Bureau of Agricultural Economics, United States Department of Agriculture, through Mr. Carlos Campbell, Mr. Leo Schaben and Mr. A. C. Edwards.

have a different curve, as cases are present throughout the year, but, lacking an actual epidemic peak, one can ignore these figures for the present. In this regard, one need only call attention to Italy, which had an endemic for two years in all months, followed by an epidemic in 1929 with the greatest incidence of cases falling in the usual epidemic months of August and September. If we except the Union of Soviet Socialist Republics, two localities are peculiar in that a great number

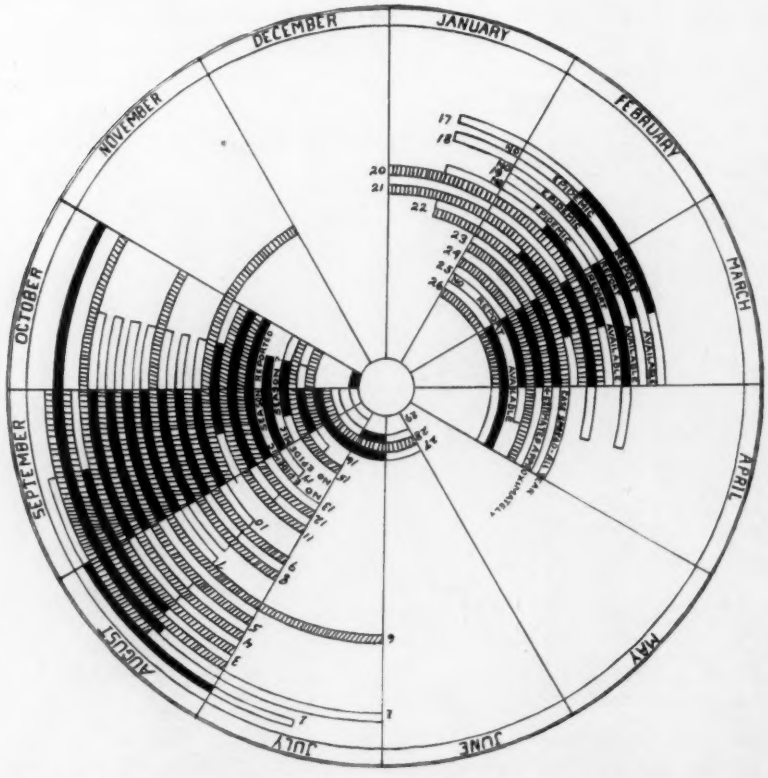


Chart 11.—Approximate vegetative season of apples in various countries, with the peak of apple harvest, compared with epidemic peaks of poliomyelitis: *1,* Spain; *2,* United States; *3,* Italy; *4,* Hungary; *5,* Australia; *6,* France; *7,* Belgium; *8,* Rumania; *9,* Germany; *10,* Denmark; *11,* Norway; *12,* Canada; *13,* Russia, *14,* Japan; *15,* Sweden; *16,* Great Britain; *17,* Ecuador; *18,* Peru; *19,* Bolivia; *20,* Paraguay and Uruguay; *21,* Argentine Republic; *22,* Brazil; *23,* South Africa; *24,* Australia; *25,* Chili; *26,* New Zealand; *27, 28* and *29,* California. The entire arc represents the total estimated harvest time; the black portion of the arc, the approximate harvest peak; the cross-barred arc, the estimated season of epidemic poliomyelitis.

of cases occur practically every month. They are Italy and California. Italy as well as California has a higher temperature and a necessarily

longer growing time. In the latter state, the grains are less cultivated, and "perishables" of all types are grown. In the epidemic of 1931 in California, there were two definite incidence peaks, as will be noted from chart 8. This would seem to be incongruous in the light of what was said previously and to contradict the statements made before, that the disease peak coincides with the harvest peak, until we remember that this state has a length of nearly 10 degrees of latitude, with four agricultural areas—the northern, southern, central and Imperial Valley districts—each with its own separate harvesting dates, which sometimes overlap. Taking apples again as our standard, we find that there are three harvesting peaks, depending on the district considered, and possibly two or three crops of small vegetations. When we study the curves and plotted lines in chart 8, we again find some similarity, though the harvesting peaks are a little late.

In Italy there is north temperate zone vegetation north of the Apennines, and were we to except the narrow Ligurian strip along the Ligurian Sea, lying in front of the Apennines and separating them from the Piedmont plain, we will find that practically all of the cases of infantile paralysis in Italy in the epidemic of 1929 occurred north of the Apennines in the area of north temperate zone vegetation. This does not mean, of course, that the disease may not occur elsewhere in Italy, since it has been reported in Naples [11] and in Rome,[15] but the explosive epidemic of 1929 occurred in the area mentioned. It was curious that in Rome the morbidity was higher in the spring and fall of the year.

COMMENT

Infantile paralysis need not necessarily be due to fruits, etc., but the "perishables" might serve as one of the vehicles by which the disease is carried from one person to another. Such a conception would explain the somewhat dispersive and explosive type of infection spread that occurs during the harvest peak. Aycock's "southern incidence curve" [7] could be explained on the basis of an earlier harvesting date.

Aycock has called attention to the fact that the incidence of this disease bears a resemblance to the incidence of typhoid. Kling [9] has noted its resemblance to dysentery. Its coincidence with gastrointestinal disease is very apparent if one constructs a dysentery percentage curve from figures found in the Japanese Sanitary District report for 1929 [16] (chart 9), with its peak in August and September. One can see the obvious similarity between the curves of the incidence

15. Cannarsa, F.: Statistical Clinical Contribution to Heine-Medin's Disease in Rome from 1901 to 1920, Pediatria **32**:215, 1924.

16. Japanese Sanitary Bureau Annual Report, 1925-1926.

of poliomyelitis and of dysentery. The typhoid curve of the same district is likewise plotted, and confirms Aycock's epidemiologic observations.

Flies are a common carrier of typhoid and probably of dysentery. In his article, "The Typhoid Toll," Johnson [17] detailed the number of flies that were caught weekly in various places of New York. The elements of chance entered a great deal into this consideration, but when the figures are arranged and a curve plotted (chart 10), it is interesting to note that the curve of the greatest number of flies occurs a little before the season of greatest incidence of the disease. However, the factors of error in this consideration are so great that definite conclusions are impossible.

The handling of infected fruit or vegetables by infected people, the eating of infected fruits and vegetables by cattle, with subsequent infection of milk, and the spreading of infection from fruit to fruit by flies—all of which could occur at the season of greatest plenitude of fruits and vegetables—could serve to explain the spread of the disease and the incidence at this time of the year. If we add to this Kling's conception of water as a possible source of infection, all the factors of the epidemiology of the disease could be explained.

The plots of our own district show that the incidence curves of the disease follow closely the plotted lines of the harvest of "perishables." The curves for other countries are inferential approximations, perhaps not quite accurate enough, but they nevertheless bear a striking degree of similarity to our own.

Figures from the League of Nation's reports [5] show that Austria, Hungary and Switzerland have experienced recent epidemics. We have no data about the "perishables" in these countries, but in Hungary the wheat harvest begins on June 1 and continues throughout July. Their first date is later by about two weeks than our earliest harvesting date in the United States. It would seem that if vegetation followed the same course as in our country, a rough approximation of the perishable vegetative season should be in August or September, approximately coincident with the peak of case incidence. The same reasoning could apply to Austria and Switzerland.[5]

No definite conclusions can be drawn, but it seems to us interesting that the peak of the incidence curve for poliomyelitis should correspond so closely to the peak of the harvesting curves in our locality. In years of great drought, temperature and precipitation would affect the crops only in a secondary manner. Perhaps this is why the first

17. Johnson, G. A.: The Typhoid Toll, J. Am. Water Works A. **3**:249 (June); 854 (Sept.) 1916.

impression of those who study poliomyelitis is that prolonged dryness seems to usher in epidemics.

The reported number of cases from Japan are few compared with the large incidence in the United States, but it also must be pointed out that the Japanese staple diet is not the same as ours and that the production of perishable fruits on a commercial scale was negligible until a few years ago.[18] The cherry tree in Japan is a symbol of beauty, not of plenitude.

Since the closeness and density of population should make the spread of cases easier, the few cases that occur in China would have to be explained.

Previously, we [19] noted that the agglutinin titer for Bacillus coli was found increased in the blood serum of patients in the convalescent stages of infantile paralysis. One might theorize that protection against this disease could be accomplished on the one hand by a high degree of immunity against Bacillus coli and its products or on the other by the possession of an actual immunity against the original factor that initiates the disease. In those districts, then, in which sanitation is primitive and the incidence of gastro-intestinal disease high, or in the places where dysentery is rampant because of geographic considerations, we might not even find the disease. This might explain the relative infrequency in China. It might also explain the relative infrequency of this disease east of the Mississippi and in the southeastern part of the United States. Of course, accurate reporting may also have something to do with the situation. Likewise, the percentage of available material may be a great factor in incidence.

CONCLUSIONS

1. In the district in and about Cleveland, the height of the incidence curve for poliomyelitis occurs at the peak of the harvest curve for the perishable fruits and vegetables.

2. Nearly similar findings are obtained in other countries.

18. Edwards, A. C.: World Apple Production and Trade, Rep. F. S. 57, U. S. Department of Agriculture, September, 1932.

19. Toomey, J. A.: Increase in Agglutinin Titer Against Colon Bacilli in Bloods of Infantile Paralysis Patients, to be published.

PREVENTING LOSS OF WEIGHT IN THE NEW-BORN

I. NEWTON KUGELMASS, M.D.
RUTH E. L. BERGGREN, M.A.
AND
MILDRED CUMMINGS, M.A.
NEW YORK

Loss of weight in the new-born is sanctioned universally. It is a period of semistarvation during the first few days of life that is too stupefying to be ignored, too debilitating to be physiologic, too prolonged to be a sacred law of nature. A century ago Clausius was the first to record the characteristic loss in weight of the new-born. And it is still being recorded, apparently without question. Civilization may have perfected the physique of the new-born, but it has simultaneously impaired the maternal secretion of milk; it may have improved the methods of delivery, but it has not been contributory in combating birth shock in the new-born. The present postnatal procedure of awaiting an ample food supply from the mother is no longer productive of the nutritional adequacy that obtained in primitive times. The modern consequence is an initial period of semistarvation, a condition non-existent among animals and bushman progeny. Several questions arise in a careful study of this problem.[1] Is the loss of weight in the new-born necessary? Is it detrimental? What is its cause? How can it be prevented? Is its prevention advantageous?

IS THE LOSS OF WEIGHT IN THE NEW-BORN NECESSARY?

The new-born infant is markedly underdeveloped for the duration of human gestation. Birth involves an abrupt change in the mechanism of the new-born infant. It is unprepared for its individualized existence in comparison with other mammals, occupying a place somewhere between the domestic mammal and the wild marsupial, whose young remain dependent on the mother. Man appears to be the striking exception in his slow rate of intra-uterine development, attaining a birth weight of about a quarter of that of other animals. Although the relation between birth weight and gestation time is elastic, it appears on an analysis of animal species that the larger the animal the longer is

From the Department of Pediatrics, the Fifth Avenue Hospital.

1. Kugelmass, I. Newton: Clinical Nutrition in Infancy and Childhood, Philadelphia, J. B. Lippincott Company, 1930.

its embryonic life. If the birth weight is plotted against the gestation time on double-log paper, a straight line relationship is found, except for a slight deviation in the case of the heaviest animals. There is some law operative which insures that these limitations necessarily follow. The only reason for using double-log paper is the convenience of getting the data on the same graph (fig. 1).

The line does not meet the coordinates at the zero point. This means that no matter how small the mammal, an appreciable time takes place for development. For a mammal as small as a gnat a surprisingly long gestation would probably be observed. For example, the mouse,

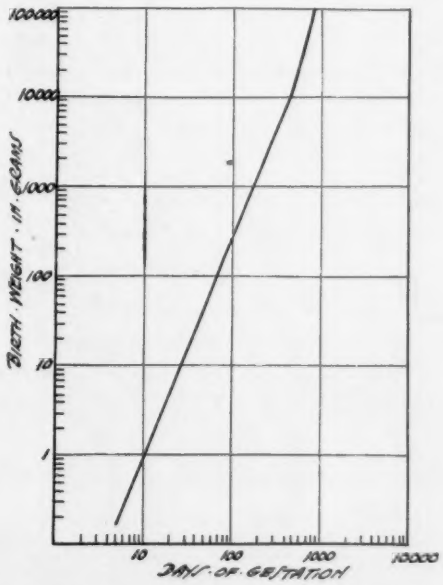


Fig. 1.—Relationship between birth weight and period of gestation.

which is 260,000 times as small as an elephant, does not have that many times as short a gestation period but only thirty-two times as short. The gnat evidently has a gestation period of about eight days. The explanation for this lies in the time required for differentiation. And the time required for the differentiation of man in utero is out of all proportion to that for all animal species. Yet the product is about a fourth of the expected birth weight because of adjustment to the human female. But this high degree of underdevelopment makes supervision of the new-born all the more urgent to meet potential pathologic changes with delicate desiderata.

The neonatal gradient of growth continues unaltered during the postnatal period. The loss in weight during the first week of life does

not reflect any deep-seated change in the growth trend of the neonatal period. The physique of the new-born infant is so labile a structure that it reacts manifestly to minor exogenous and endogenous factors that begin to play on it after birth. But there appears no fundamental change in the growth trend which proceeded undisturbed in utero. This fact we have felt necessary to establish in order not to attempt to alter what was once considered a transitional depression in the growth process.

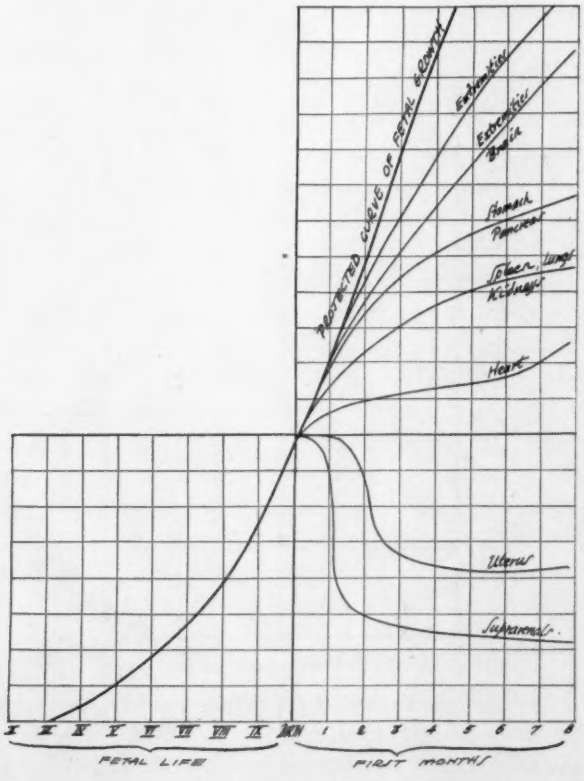


Fig. 2.—Growth in prenatal and postnatal life: The simple type of growth curve of the fetus becomes differentiated into various types of growth during infancy, but not during the transition period following birth. (From Scammon.)

Scammon [2] demonstrated the various types of growth that occur in the body as a whole in comparison with the changes in size of its tissues and organs. They fall into approximately four types: the general type exemplified by the relative growth of the body as a whole, the genital

2. Scammon, R. E., and Calkins, L. A.: The Development and Growth of the External Dimensions of the Human Body in the Fetal Period, Minneapolis, University of Minnesota Press, 1929.

type exemplified by the relative growth of the genital organs, the neural type exemplified by the relative growth of the brain and nervous system, and the lymphoid type exemplified by the relative growth of the lymphoid organs.

An analysis of the growth of the body and its organs in the fetal period reveals that the course of growth of all is essentially the same. Each organ as well as the body as a whole shows a period of slow

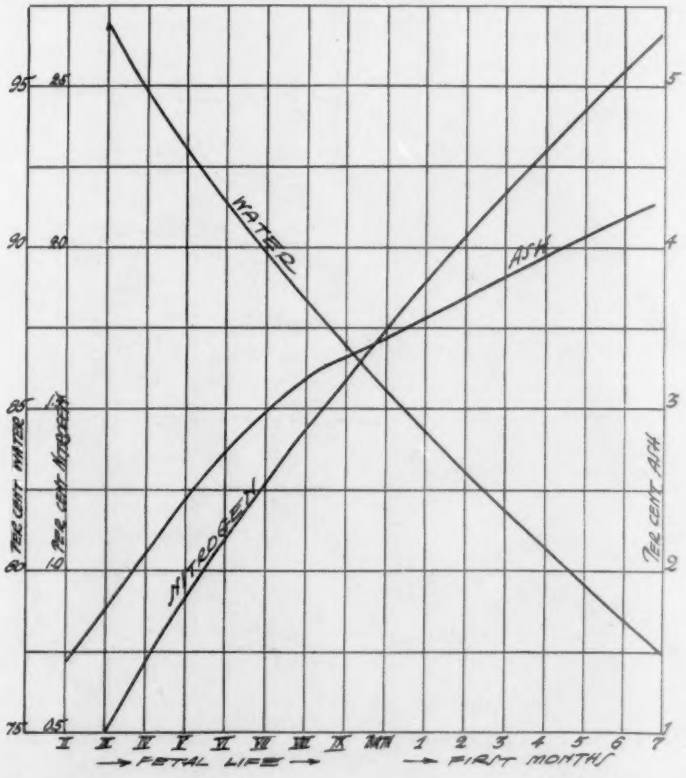


Fig. 3.—Chemical changes in prenatal and postnatal life. (From Moulton.)

growth early in the fetal period, a rather marked change in rate about the middle of the fetal period and a rapid growth thereafter. If one compares this prenatal growth with the postnatal growth, by bringing them together on an equivalent scale, it becomes evident that the characteristic form of growth of the fetal period continues for several months after birth. This transition is illustrated schematically in figure 2.

At birth the uterus and suprarenals alter from the typical trend; then the rate of growth of the heart and viscera follows another course; then the stomach and pancreas assume a slower rate; then the rate of

growth of the nervous system drops away from the fetal curve, and finally the trunk and extremities deviate. But the transition from the uniform rate of growth, so striking in the fetal period, to the diverse character of extra-uterine life is not simultaneous with birth. The transition to infantile increments of growth types takes place very slowly after birth and in different parts of the body at different times during infancy. The resultant change in ponderal growth is negative so far

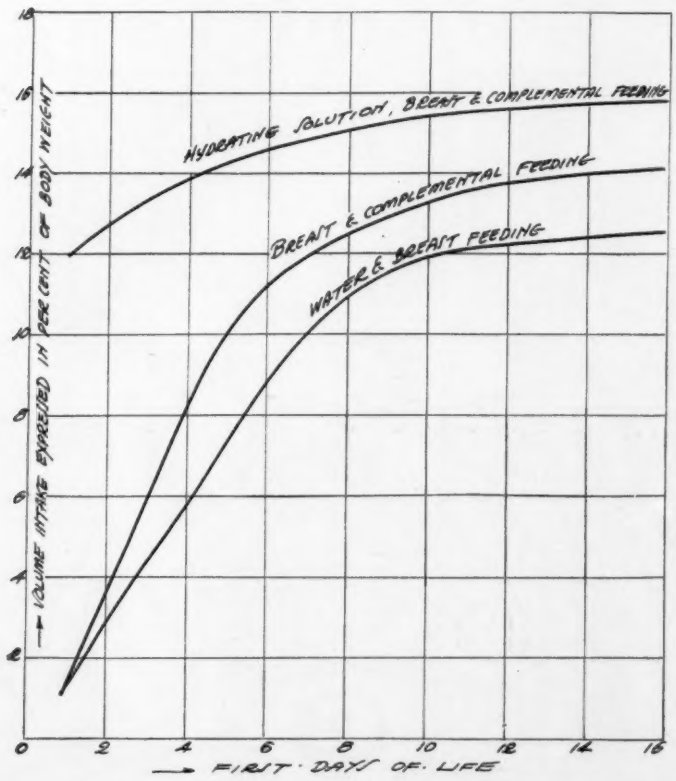


Fig. 4.—Total intake of hydrating solution as compared with total fluid intake of controls, expressed in percentage of body weight.

as it can reflect itself in the weight of the new-born baby during the first weeks of life. Therefore the loss in weight during the first days of life is unrelated to the deep-seated changes that proceed throughout infancy. There is no abrupt change in the growth patterns of the body and its organs immediately after birth. The loss in weight of the new-born infant is obviously exogenous in nature.

The continuity of deep-seated chemical changes proceeds throughout the transition from prenatal to postnatal life. Moulton [3] was the first

3. Moulton, C. R.: J. Biol. Chem. **57**:79, 1923.

to pursue the chemical course of the whole life span of various species with regard to the water, nitrogen and ash content. He demonstrated that the composition at birth is unrelated to any of the life variables. In other words, the act of birth or hatching throughout the animal species is unimportant so far as the fundamental mechanisms of growth are involved. Embryologists have thus far concluded that the time at which birth takes place in the life span of the animal is related only to the adaptations due to different modes of animal life. The underlying processes of physicochemical maturation remain unaltered. Figure 3

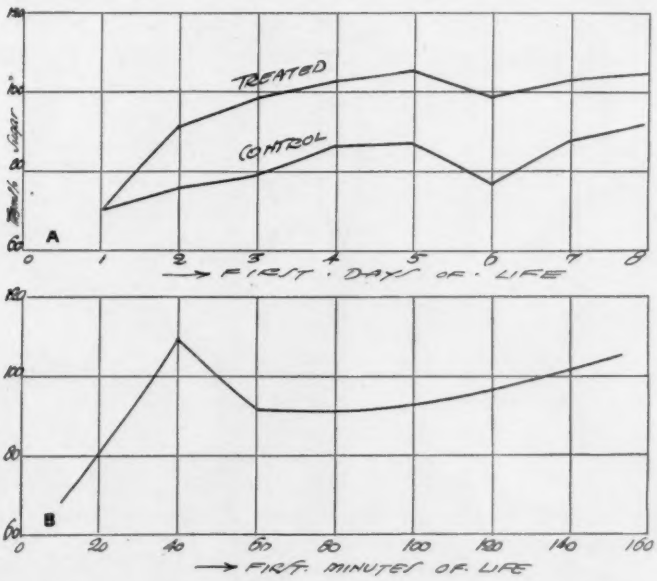


Fig. 5.—*A*, blood sugar curves of new-born infants. *B*, sugar tolerance curve of new-born infants.

shows the continuity of the composition of the new-born infant in water, nitrogen and ash, undisturbed by the birth process. The postnatal changes observed are therefore beyond question environmental, and necessarily rest on the pediatrician for alleviation.

Postnatal loss of weight does not obtain in animals. The birth process appears to be unrelated to any change in the cycle of growth postnatally. This has been the observation of Scammon,[4] Jackson [5] and

4. Scammon, R. E.: Proc. Soc. Exper. Biol. & Med. **20**:102, 1922.
5. Jackson, C. M.: Am. J. Anat. **9**:118, 1909.

others for man and of Donaldson,[6] Lowrey,[7] Schmalhausen [8] and others for animals. An inquiry into change of weight in animals after birth or hatching shows that no losses result anywhere comparable to those observed in man. Animal species of all sizes, of varied gestation periods and of all scales in evolution, receiving no scientific supervision after birth or hatching, appear to thrive either immediately or, at the utmost, after the second day of extra-uterine life. These are the results accumulated from data obtained from animal husbandmen of experimental stations and from curators of zoological parks in this country and abroad (table 1).

This survey of uninterrupted growth after birth of animal species is a revelation of the unique place that man holds in his adjustment to environmental forces from the very moment of birth. The transient

Fig. 6.—Capillary tube used in determination of refractive indexes: *A*, level of blood; *B*, boundary line of serum.

loss in animals is truly physiologic, in that it consists of some skin, hair, amniotic fluid, meconium and urine. The changes represent the irreducible minimum of loss in weight, which lasts from an hour to a day, since maternal food is already available and adequate for the young. Animals seek the breast immediately after birth and suck continuously unless interrupted by sleep. Even the bushman mother, free from the stress and strain of modern life, is able to nurse her offspring in sufficiency following birth, thus minimizing civilization's lag in growth after human birth.

---

6. Donaldson, H. H.: Tr. 15th Internat. Cong. Hyg., 1912, vol. 2, p. 410.

7. Lowrey, L. G.: Am. J. Anat. **12**:107, 1911.

8. Schmalhausen, J.: Arch. f. Entwcklngsmechn. d. Organ. **109**:455, 1927; **110**:33, 1928; **113**:462, 1928; **115**:668, 1929; **123**:153, 1930.

Not only are animals provided with nutritious milk immediately after birth, but they are physiologically able to utilize it. The foal may almost be said to be born at a gallop, for it is on its feet in thirty minutes, obtaining an abundance of rich milk. It is generally thus with ruminants, for they are more mature at birth than is a child at 2 or 3 years of age. New-born solipeds lose no weight, for within an hour after birth they consume up to 5 per cent of their weight in milk. New-born carnivora are locked at once to the teat, and suck continuously until

TABLE 1.—*Growth Values of Animals*

Animal	Gestation Time, Days	Birth Weight, Gm.	Loss of Birth Weight, per Cent	Duration of Weight Loss, Hours	Average Daily Gain, Developmental Period
Opossum	8	0.5	0	0	....
Kangaroo rat	8	0.5	0	0	....
Mouse	21	1.6	..	..	0.08
Rat	21	5	2	6	0.24
Marmot	21	25	..	..	1.2
Rabbit	30	70	0	0	....
Cat	60	100	0	0	....
Wolf	61	250	0	0	4.0
Guinea-pig	63	120	2	24	2.0
Puma	92	500	0	0	5.5
Lion	105	1,000	0	0	10.0
Pig	120	1,700	3	6	14.0
Lemur	144	240	0	0	....
Goat	150	2,800	3	12	24.0
Sheep	150	4,000	4	12	26.0
Mandrill	200	480	0	0	....
Macaco	200	480	0	0	....
Hippopotamus	225	50,000	4	48	200.0
Bear	240	1,500	0	0	7.4
Eland	250	60,000	0	0	....
Stag	250	24,000	3	24	100.0
Chimpanzee	260	1,000	4	24	....
Roe-deer	280	3,000	0	0	11.0
Cow	285	37,000	3	24	125.0
Horse	330	50,000	1	24	200.0
Seal	350	10,000	0	0	30.0
Camel	360	80,000	1	74	400.0
Donkey	370	20,000	1	24	53.0
Rhinoceros	510	50,000	4	48	200.0
Elephant	615	240,000	3	48	400.0

sleep overcomes them. Even if they are born out of doors during the winter, the covering with nonconducting hair reduces the loss of heat to a minimum. The young occasionally lose the tips of the ears or the tail if the temperature is extremely low, but they take such an abundance of food immediately after birth and assimilate it so well that no loss in weight occurs. The loss of weight in the new-born infant is unnecessary when compared philogenetically with similar birth processes in the entire animal kingdom.

WHAT IS THE CAUSE OF LOSS OF WEIGHT IN THE NEW-BORN?

Universality of loss of weight in the new-born without exception has glorified the phenomenon into a so-called physiologic law. A century of literature acknowledges the existence of varied attempts at physio-

logic correlation. Attempts at interpretation of the loss in weight in the new-born have led early investigators far afield. Landois [9] maintained that early ligation of the umbilical cord was preventive of loss of weight. Porak [10] related the loss of weight to the icterus, and maintained that the two phenomena are inseparable. Neither of these observations has gleaned any physiologic rationale, and has never been confirmed theoretically or clinically. Other workers sought an explanation of the loss of weight in the immaturity of the new-born infant's digestive organs.

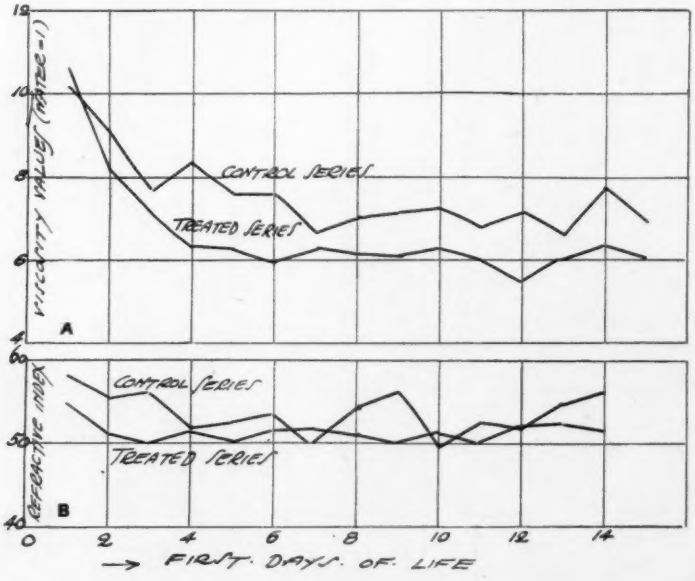


Fig. 7.—*A*, curves of viscosity of blood. *B*, curves for refractive index of blood.

Such elusive statements are more apparent than real, for they cannot be substantiated by observations on new-born infants. Some observers have seriously reported increased urinary secretion with late ligation of the cord as a factor contributory to the excessive loss in weight. Some have even related the loss of weight in the new-born to the degree of development of the mammary mechanism because new-born infants of multiparas lose less than those of primiparas. Some investigators have placed the responsibility on extrinsic factors to which

9. Landois: Monatschr. f. Geburtsh. u. Gynäk. **22**:194, 1905.
10. Porak, C.: Rev. mens. de méd. et chir. **2**:334, 1878.

the new-born must become adjusted. Bakwin [11] found no correlation between the loss in weight during the first days of life and the nursery temperature, humidity or wet bulb depression. Careful clinical observers, however, have for a long time maintained that the early inadequate supply of breast milk, the infrequent feedings and the wholesome neglect of the new-born are the factors responsible for the loss in weight. All incidental observations are figments of imagination unrelated to the actual problem.

The excretion of the new-born is neither massive nor unusual, for the sum total of loss due to swallowed amniotic fluid, vernix caseosa,

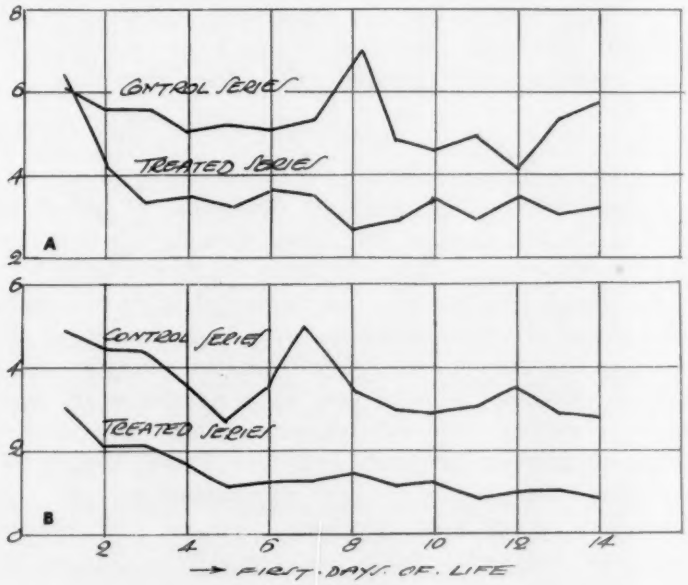


Fig. 8.—*A*, curves of clotting time. *B*, curves of bleeding time.

meconium and urine is negligible in comparison with the actual loss of weight during the first days of life. Cammerer [12] demonstrated metabolically that the loss in weight is nothing more unusual than the difference between intake and output. The greater portion of the output is independent of alimentary elimination. It consists of loss of food from the skin and lungs as insensible perspiration, not due to an excessive metabolic rate, but rather to an inadequate total intake of fluid to compensate for the daily requirement. Actually, therefore, the loss in weight is due to some starvation, which is never physiologic.

11. Bakwin, H., and Bakwin, R. M.: Am. J. Obst. & Gynec. **18**:863, 1929.
12. Cammerer, W.: Jahrb. f. Kinderh. **53**:381, 1901.

Benedict and Talbot [13] measured the daily output of heat in resting new-born infants as 170 calories in twenty-four hours. This caloric intake is rarely if every achieved by the ingestion of colostrum. Bailey and Murlin [14] calculated the caloric value of colostrum to be 65 calories per hundred cubic centimeters, thus necessitating a daily caloric requirement of 270 cc., or over 1 ounce (28.3 Gm.) in eight nursings, an amount which the modern human breast does not secrete until the fourth day. Not only is the caloric intake inadequate when the new-born infant is allowed to depend on the secretion of the breast during the first days of life, but the total fluid requirement, far more indispensable than mere caloric fulfilment, is never thus maintained. Metabolic studies of the new-born infant reveal a striking inadequacy in fluid and caloric intake during the first days of life, a loss in weight being a necessary consequence of this initial period of semistarvation.

IS THE LOSS OF WEIGHT DETRIMENTAL IN NEW-BORN INFANTS?

Loss of weight in the new-born is due to starvation. It is never beneficial at any time during infancy or childhood. It cannot well be indicated in the first days of life, which abound with possibilities of pathologic conditions. Postnatal starvation is not resorted to for therapeutic purposes, but is rather the consequence of sanctioned neglect. A three day period of semistarvation may well be a procedure of choice in combating a derangement more detrimental than starvation per se can produce. Such therapy is never ideal, but is a choice of the lesser evil. It cannot be gleaned from our previous comment that there is any rationale for starvation during the first days of life. We do not mean to imply that a balanced dietary is at all necessary once the fetus has been deprived of maternal nutrients. We do wish, however, to emphasize the basic need, during the first days of life, of fluid to facilitate the transitional metabolic changes incident to birth and of carbohydrate, not only to maintain the basal heat expenditure, but to overcome the hypoglycemia secondary to birth shock.

Physiologic trauma of birth is conducive to the development, to some degree, of what we call "birth shock." We are adducing evidence that the clinically characteristic behavior after birth—somnolence, stupor, lethargy and anorexia—are manifestations of shock precipitated not only by the traumatization but by an increased activity of the sympathetic nervous system, which has the effect of lessening the volume of plasma. Therefore it appears wise not to accept these physiologic

13. Benedict, F. G., and Talbot, F. B.: Physiology of the New-Born Infant. Character and Amount of Katabolism, Washington, D. C., Carnegie Institution, 1915, publ. 223.

14. Bailey and Murlin: Am. J. Obst. **71**:526, 1915.

symptoms but rather to alleviate them by the diligent administration of at least fluids. Modern delay in the early availability of a supply of secretion from the breast no longer warrants dependence on this natural source in the spirit of the primitive state. While the detriment to the new-born is only partial or even slight without the addition of early artificial feeding, the consequences of semistarvation are cumulative toward the end of that period.

Grulee [15] has clearly pointed out how difficult the path of the new-born infant is, beset with a host of enemies still unique and obscure—toxic symptoms with lethargy, inanition fever, vomiting, diarrhea, jaundice, impetigo, etc. These so-called physiologic manifestations as a part of the new-born infant's transition to his new environment are markedly aggravated if he is allowed to persist during the first days in a state of semistarvation. Tyson [16] made a careful analysis of the infants in his new-born service. He observed 10 per cent with fever after the second day of life, but half of them had been suffering from frank manifestations of dehydration, which cleared up within twenty-four hours on a more ample administration of fluids. Dehydration is not readily recognized until toxic symptoms supervene. Meconium persists with resultant unfavorable absorption unless a sufficiency of fluid makes its early elimination possible. Infectious invasion is more prevalent in inadequately hydrated tissues. Adequate administration of fluid from birth on contributes greatly to the functional improvement of the new-born in the prevention of secondary disturbances.

HOW CAN THE LOSS OF WEIGHT BE PREVENTED?

The effectiveness of therapeutic procedure parallels its degree of dependence on the physiologic changes which are to be corrected. Many attempts have been made to decrease the loss of weight in the new-born, but without consistent results because the methods devised were apparently unrelated to the fundamental cause of the sudden drop in body weight. Benestad [17] and Altheer [18] in Germany, Krueger [19] and Ingerslav [20] in Sweden and others have erroneously assumed that the loss in weight was the result of an adjustment to the new-born infant's first food intake. They maintained that the loss was inevitable because of the

15. Grulee, C. G., and Bonar, B. E.: The Newborn, New York, D. Appleton and Company, 1926.

16. Tyson, R. M.: Arch. Pediat. **45**:21, 1928.

17. Benestad, G.: Jahrb. f. Kinderh. **80**:21, 1914.

18. Altheer: Ueber regelmassige tagliche Wagungen der Neugeborenen, Dissertation, Basel, 1874.

19. Krueger: Arch. f. Gynäk. **7**:27, 1875.

20. Ingerslav, E., quoted by Berger, C. J.: Fragen über die Geburt des Menschen und die Geburtshilfe, Copenhagen, J. Frimodt, 1910, p. 101.

immaturity of the alimentary organs and hence their inefficiency in adequate utilization of the first food offered. Such an assumption is unwarranted even in the premature infants, and of course is nonconducive to any rational procedure for preventing loss in weight. These observers went so far as to place new-born infants at the breasts of mothers whose own babies had been born several days previously, but the new-born infants who nursed from an abundant supply lost more weight than the controls.

McIlroy,[21] in England, assumed that the loss of body heat in the new-born was largely responsible for the loss of weight, and so

TABLE 2.—*Observations on the Initial Loss of Weight in New-Born Infants*

Author	Weight, Gm.	Loss of Birth Weight	Per Cent of Infants Regaining Birth Weight on			Prelacteal Procedure
			7th Day	14th Day	21st Day	
Adair	266	8.0	..	..	..	Colostrum and water
Autonov	...	...	6	50	75	Colostrum and water
Bakwin	...	8.0	..	..	..	Colostrum and water
Bauchaud	200	...	..	..	..	Colostrum and water
Bergman	...	...	11	..	..	Colostrum and water
Borrino	240	...	..	..	..	Colostrum and water
Dluski	212	...	..	..	..	Colostrum and water
Drossel	...	8.8	..	..	..	Colostrum
	...	7.5	..	..	..	Water
	...	7.5	..	..	..	10% sugar
	...	6.8	..	..	..	Ringer's solution
Eder	...	5.0	67	83	..	Sodium citrate-lactose solution
Fuhrman	...	9.4	..	..	..	
Gregory	203	...	..	..	..	Colostrum and water
Heiman	...	...	..	..	80	Colostrum and water
Herzfeld	...	9.8	..	..	..	Colostrum and water
Kirstein	...	7.8	..	..	..	
Neff	...	7.5	..	85	..	
Oberg	...	...	10	80	..	Colostrum and water
Randall	...	6.1	..	90	..	Complemental feeding
Robertson	330	9.2	..	..	..	Colostrum and water
Schulte	...	...	..	45	..	Colostrum and water
Schulz	...	...	..	50	..	Colostrum and water
Seifert	...	...	20	..	..	Colostrum and water
Steiner	222	...	..	..	..	Colostrum and water
Tyson	266	8.1	..	62	..	Colostrum and water

attempted to diminish it by resorting to continuous oil baths for maintaining body heat at a maximum. Lascano and Halac,[22] in Italy, approached the problem on the basis of eliminating the initial starvation period of the new-born infant by early nursing. Drossel [23] forced the fluid intake the first few days to overcome dehydration, and was able only to reduce the average loss of weight of 8.8 per cent to 7.5 per cent, with a diminution in deficit of regained birth weight of 3.6 per cent in the former as compared with 2.5 per cent in the latter group on the tenth day of life. The administration of Ringer's solution reduced the loss in weight to 6.8 per cent, and complemental feeding to 5.9 per cent.

21. McIlroy, A. L.: Brit. M. J. **1**:67, 1925.
22. Lascano, J. C., and Halac, E. S.: Rev. de especialid. **3**:848, 1928.
23. Drossel: Arch. f. Kinderh. **88**:30, 1929.

Eder and Bakewell [24] have decreased the initial postnatal loss in weight to an average of less than 6 per cent by the routine administration of 5 per cent lactose solution containing sodium citrate. They were thus able to obtain an earlier regain of birth weight in most of the new-born infants and a marked diminution in the number of cases of inanition fever (table 2).

We have initiated our present study as a result of favorable experiences in the nutritional care of premature infants. During the first days of life we were able to correct the striking physiologic deviations from the norm by the oral administration of 5 per cent dextrose solution reenforced with 5 per cent glycocoll every two hours immediately after birth. The dextrose consistently alleviated the low blood sugar observed in these premature infants, and the glycocoll appeared to raise the internal body heat to a normal level, thus overcoming the low birth temperature. The continuous administration of the solution between feedings the first two days after birth prevented in a large measure the initial loss in weight, which the premature infants could not well afford. This observation in connection with premature, debilitated and small infants led us to a more careful study for the development of a solution which would be preventive of the initial loss in weight in all new-born infants.

We have studied the relative merits of complemental feeding immediately after birth with the preliminary administration of a solution for the alleviation of birth shock. We have adduced evidence of the existence of the latter phenomenon, and have found it advantageous to offer a solution to combat the shock the first two days and then offer complemental feeding with whatever breast milk is available.

THE SOLUTION TO COMBAT BIRTH SHOCK

The new-born infant's nutritional requirement can be fulfilled only when the symptoms consequent on the physiologic trauma of birth have been alleviated. We have observed that nutritional therapy is specifically indicated the first two or three days of life, before the required normal feeding regimen is well tolerated. The actual intake of colostrum is small, even if valuable, and the ingestion of a feeding formula minimal under the most favorable conditions. The amount of food consumed in terms of percentage of body weight is less than 1 per cent on the first day, gradually increasing to over 10 per cent after the fourth day. This limited intake is a consequence of birth shock. We have therefore found it advisable to prevent initial loss in weight, not by forcing feeding mix-

24. Eder, H. L., and Bakewell, B.: Effect of Sodium Citrate on the Loss of Weight in the New-Born Infant, Am. J. Dis. Child. **42**:1079 (Nov.) 1931.

tures from the day of birth, but rather by the administration of a solution suited to physiologic needs postnatally. After a series of attempts to determine the relative effectiveness of various measures we have found the following solution the most desirable for reducing the initial loss in weight of the new-born to the irreducible minimum: gelatin (p_H 6.2), 6 per cent; dextrose, 3 per cent, and sodium chloride, 0.5 per cent. It so happens that our favorable clinical observations with this solution have brought out its identity with the gross content of colostrum. This solution was administered in consecutive staff cases immediately after birth and every two hours thereafter for the first three days, when breast milk was supplemented by a feeding formula according to the needs of the individual infant.

The new-born infant's requirement of the constituents of this solution may be envisaged from the following considerations. Continued administration of water is indispensable from birth on. Stander and Tyler [25] found that the water content of the blood of the fetus was less than that of the mother. We have observed, by determination of the refractive index, viscosity and volume of blood, that the blood is concentrated at birth as a consequence of shock and subsequent deprivation of water. Normally, the water content of the new-born infant is about 85 per cent, gradually diminishing with age. This high degree of tissue hydration at birth is indicative of the correspondingly high degree of immaturity of the body, and necessarily bespeaks the tremendous water requirement at this stage of life. The high degree of bound water content in the new-born is indicative of its indispensability in the body's immutable structure. This presupposes no available water reserve for ready mobilization comparable to the fat or carbohydrate supply.

Determination of the amount of water eliminated from the lungs and skin of new-born infants revealed it to be about three times as great per kilogram of body weight as that of an adult. In fact, the adult retains about 35 Gm. of water per kilogram of body weight, while the new-born infant binds about 150 Gm. of water per kilogram of body weight, or over four times the adult requirement. These considerations of the water metabolism emphasize the dire need for the continued administration of fluid immediately after birth. An inadequate fluid intake in the new-born infant is conducive to the development of a variety of symptoms, initially alimentary in nature, long before frank dehydration becomes apparent to the clinician. Mere water intake, however, has shown no consistent maintenance of the required tissue hydration in the new-born. The marked lability in water metabolism is apparent from hourly determinations of the refractive index of the blood

25. Stander and Tyler: Surg., Gynec. & Obst. **31**:276, 1920.

in comparison with that of older infants. We have therefore offered not only water but other dissolved constituents to favor its retention. This instability in water metabolism has been interpreted on the basis of immaturity of the central water-regulating mechanism, but we have found no evidence for such an assumption.

Addition of nutrients for stabilizing the water intake in new-born infants has been adequately met by maximal amounts of gelatin and minimal amounts of sodium chloride. Gelatin has several physiologic advantages for the new-born: It is markedly hydrating, well tolerated and easily assimilable. Sodium chloride, a neutral salt, prolongs the retention of ingested water by virtue of the dual properties of the salt—the hydration of the sodium ion and the neutral salt effect. Studies of the composition of the blood in the new-born have shown that the total protein content is at low normal levels in comparison with older children, a further indication for the addition of gelatin. Similarly, the sodium chloride content of the blood of new-born infants has been observed to be a low normal level, gradually rising with a regain of the birth weight. Hence the justification for the addition of minimal amounts of sodium chloride to raise the low concentration of plasma sodium chloride. This restores to blood and tissues their normal degree of hydration in view of the absolute relation of this salt to the water content of the body. But the addition of gelatin has another advantage. By virtue of its specific dynamic action, it elevates the body temperature, which is subnormal at birth. And its metabolic products likewise contribute toward the elevation of the blood pressure, lowered as a consequence of birth shock.

Determination of the blood sugar at birth reveals a consistently low normal concentration. Therefore dextrose has been added to the solution offered the new-born, not only to maintain a normal blood sugar level but to offer a readily assimilable carbohydrate adequate for the basal metabolic needs of the body during the first days of life. The entire solution is isotonic, because half of the molecular concentration consists of 3 per cent dextrose and the other half of 0.5 per cent sodium chloride. The solution is further made colloidally osmolar by the addition of 6 per cent gelatin, which parallels the concentration of the blood. The consistency of the solution, which is warmed before feeding, is such as to favor retention, thus overcoming the regurgitation so prevalent in young infants.

The tryptic rather than peptic digestion of gelatin favors a slowness of molecular absorption from the solution, advantageous in the initial alimentary adjustment of the new-born. Incidentally the gross concentration of the solution parallels that of colostrum, although our purpose in its administration is not that of nature in providing the new-born with colostrum. The great emphasis that has been placed on the

physical and chemical properties of breast milk so far as its universal tolerance in infants is concerned has led to its apparent simulation in artificial feeding mixtures. And so it is in that spirit of simulation of gross properties rather than in duplication of the minutiae of colostrum that we compare the solution herein devised. We have attempted to modify the solution further for its acid-base content because of the low p_H observed during the first day of life and the reciprocal carbon dioxide content that parallels the so-called "acidosis of the new-born." The variability of the acid-base equilibrium in the new-born and the refinements that we found necessary were out of all proportion to the disturbances produced by the attempted administration of such a modified solution. We have therefore been content with a solution which will maintain the required level of tissue hydration and be well tolerated, nonirritating and inexpensive.

Preparation of the Hydrating Solution.—To a cup of cold sterile water add about 6 level tablespoonfuls of gelatin (p_H 6.2), about 3 level tablespoonfuls of dextrose and about 1 level tablespoonful of table salt. The gelatin should be free from flavoring, coloring and sweetening if it is to be well borne as the first nutrient of the new-born. Allow mixture to soak for five minutes. The remaining 3 cupfuls of water required to make a quart (946 cc.) of the solution are brought to the boiling point and added slowly to the mixture. This nutrient solution for the new-born consists of 6 per cent gelatin, 3 per cent dextrose and 0.5 per cent sodium chloride, having a caloric value of 12 calories to the ounce.

CLINICAL COURSE OF TREATED NEW-BORN INFANTS

Solutions of varying composition were tested clinically for relative effectiveness in preventing loss of weight in the new-born. The soluble nutrients were selected, as previously described, on the basis of the therapeutic needs of the new-born after experiencing physiologic trauma incident to birth. Our studies of nutrition of the new-born indicated an initial requirement in a hydrating solution of a caloric value sufficient to maintain basal needs during the first three days of life. Such a solution, consisting of well tolerated substances—gelatin, sugar and salt—brought more rapid recovery from the effects of the birth shock than did the immediate feeding of milk mixtures. In fact, the final, most effective solution offered during the first three days of life actually prepared the infants for more efficient nursing and utilization of feedings. The solution was given every two hours throughout the twenty-four hour cycle. The results are expressed in table 3.

The first orientation study was to administer 5 per cent gelatin and 5 per cent dextrose between nursings during the day. Each of the fifteen new-born infants studied was put to the breast six hours after birth. The results showed an average loss of 7.6 ounces (215 Gm.), equivalent to a loss of body weight of 7.2 per cent. Although this showed some improvement over the controls, it pointed the way to fur-

ther modification of the procedure. All the new-born infants took the solution readily without force, and tolerated it equally well. Offering fluids between feedings resolved itself into four hour intervals, which proved too infrequent for fluid and caloric intake adequate to maintain the basal requirement of new-born infants.

The second solution consisted of 3 per cent gelatin, 3 per cent dextrose and 0.5 per cent sodium chloride because an increase in the salt necessarily required a diminution in concentration of the other constituents to maintain osmolar properties. This mixture, offered

TABLE 3.—*Effectiveness of Various Solutions in Preventing Loss of Weight*

Solution	Case	Schedule	Initial Loss, Oz.	Loss, per Cent	Day That Birth Weight Was Regained	Infants Gaining Weight, per Cent
Gelatin 5%, dextrose 5%.....	15	Q. 4 h. × 4	7.6	7.2	7	78
Gelatin 3%, dextrose 3%, sodium chloride 0.5%.....	17	Q. 2 h. × 6	4.7	3.9	6	76
Gelatin 3%, dextrose 3%, sodium chloride 0.5% (p_H 7.8).....	8	Q. 2 h. × 6	9.8	8.1	10	60
Gelatin 3%, dextrose 3%, sodium chloride 0.5%, sodium phosphate U.S.P. (p_H 7.8).....	12	Q. 2 h. × 6	6.2	4.8	5	92
Gelatin 6%, dextrose 3%, sodium chloride 0.5%.....	12	Q. 2 h. × 6	4.7	3.4	6	96
Gelatin 6%, dextrose 3%, sodium chloride 0.5%.....	29	Q. 2 h. × 12	2.1	1.7	5	100

TABLE 4.—*The Intake of Hydrating Solution as Compared With Fluid Intake of Controls* *

	Day of Life													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Intake of solution.....	12.0	13.5	14.0	13.5	13.8	14.5	14.5	15.0	15.5	15.5	15.8	16.0	16.3	16.5
Intake of breast milk and complemental feeding.....	1.0	4.0	7.0	9.0	10.5	11.0	12.0	12.5	13.0	13.0	14.0	13.5	14.0	14.5
Intake of water and breast milk.....	0.5	3.0	4.5	6.0	8.0	10.0	10.5	11.0	11.8	12.0	12.3	12.5	12.5	13.0

* Expressed as percentage of body weight.

every two hours during the day, reduced the loss to 3.9 per cent in comparison with the controls.

Observations of the acid-base equilibrium on the first day of life confirmed a compensated acidosis first recorded by Ylppö. Attempts at modifying the hydrating solution to contain buffered salts for treatment of the acidosis were not very satisfactory in preventing the initial loss in weight. The addition of sodium hydroxide by titration to p_H 7.8 aggravated the loss in weight and the frequency of stools. Buffered salt additions as phosphate mixtures to the same p_H produced no favorable effect on weight.

The final hydrating solution found effective clinically consisted of 6 per cent gelatin, 3 per cent dextrose and 0.5 per cent sodium chloride. When offered to twelve infants with controls at two hour intervals

during the day, the loss in weight was no more than 3.4 per cent. But when the same solution was offered throughout the twenty-four hour cycle at two hour intervals, both between and immediately after nursing, the loss in weight was the irreducible minimum, 1.7 per cent. The caloric intake exceeded the basal requirement in most new-born infants, and thus tided them over the first days of reaction to the birth process. All the infants began to gain weight on the fifth day of life at a rate which far exceeded that of the controls.

Complemental feeding in three hundred control infants merely reduced the initial loss in weight from the usual 10 per cent to from 6 to 9 per cent. The new-born infants were put to the breast six hours after birth and then continued on four-hour nursing periods with complemental feeding whenever indicated. The actual amounts offered to each baby in varying types of milk mixtures depended on the adequacy of the breast supply. The feedings were offered only after the breast

TABLE 5.—*Initial Loss of Weight in Three Hundred New-Born Controls (Breast and Complemental Feeding)*

Birth Weight	Number of Cases	Average Initial Loss, Oz.	Average Loss of Birth Weight, per Cent
9	21	11.0	8.8
8	75	9.6	7.1
7	115	8.0	6.8
6	64	6.2	6.8
5	19	6.5	6.9
4	8	5.9	6.2

supply was exhausted and continued until the total ingestion of human milk fulfilled optimal requirements in each baby. Like other observers we have adjusted the complemental feedings without endangering the baby's natural source of nutrition.

The old dictum of Czerny's, that of not offering new-born infants any feeding until the breast milk has become available, no longer holds. The purpose of that practice was to obviate the development of unnatural and harmful fecal flora, a condition that has never been confirmed bacteriologically. The modern prolonged interval for the development of an adequate supply of breast milk no longer warrants the practice of semistarvation. As a result the enfeebled infants lack the energy to nurse, particularly from engorged breasts. Incomplete emptying of the breast is in itself the most effective stimulus for the secretion of milk. The consequence is a vicious cycle with an unnecessarily prolonged adjustment to the optimal nutritional requirement.

Early complemental feeding invigorates the new-born infant to the extent of obtaining more breast milk; hence it is mutually beneficial in accentuating the most effective stimulus to an increased flow of breast milk. Careful regulation of complemental feeding never minimizes

effective nursing, but complemental feeding in the three hundred new-born infants observed did not result in eliminating the initial loss in weight. Administration of the hydrating solution the first three days between nursing and then complemental feeding with nursing evidently prepared the new-born infant for rapid adjustment to the required breast and artificial feeding.

The control series revealed a greater loss for larger new-born infants. Even on complemental feeding, 9 pound (4 Kg.) infants lost, on the average, about 9 per cent of their birth weight, while smaller

TABLE 6.—*Nutritional Study of Twins*

Twin	Sex	Birth Weight	Birth Temperature, F.	Initial Loss, Oz.	Loss of Weight, per Cent	Day That Birth Weight Was Regained
Treated	Male	6 lbs. 3 oz.	98.8	2.0	2.0	4
Control	Female	6 lbs. 3 oz.	98	5.0	5.0	Not regained in 14 days

TABLE 7.—*Effect of Order of Birth on Initial Loss of Weight*

Order of birth	1	2	3	4	5	6
Average loss of birth weight, per cent.	5.7	4.1	3.9	3.8	3.8	4.0
Number of cases	41	26	21	16	11	18

TABLE 8.—*Effect of Nationality on Initial Loss of Weight*

Nationality	German	American	Italian	Porto Rican	Irish	Jewish
Average loss of birth weight, per cent	3.5	4.98	4.93	5.12	5.26	5.30
Number of cases	12	12	33	12	30	40

infants lost less but never below 6 per cent of their birth weight (table 5). The striking advantage of the administration of the hydrating solution was to equalize this variation in postnatal loss of weight to the extent that the average was less than 2 per cent. Other intrinsic variations in new-born infants persisted in spite of the initial care and concern of their nutrition.

The babies in private cases outweighed those in the ward and tended to lose less after birth. Male infants consistently lost more than female infants. First-born infants tended to lose more than siblings (table 7), and in a cosmopolitan nursery differences in nationalities made their imprint even in the degree of postnatal loss in weight (table 8). New-born infants of various nationalities may be grouped in the following order in terms of the minimal loss in weight: German < American < Italian < Spanish < Irish < Jewish.

The clinical course of twins was particularly instructive in determining the value of the hydrating solution. Twins of approximately the same birth weight, varying 1 ounce (28.3 Gm.), offered an opportunity for testing the merits of the solution in such a pair, in whom intrinsic differences were as yet not manifest. Since nature apparently has difficulty in preserving the integrity of male infants in comparison with female, the female twin was selected as the control. She was offered a complemental feeding of evaporated milk mixture, 20 calories to the ounce at three hour intervals immediately after birth. The male twin was offered the hydrating solution at two hour intervals after birth for the first three days and then the same milk mixture as the control twin. Both were nursed at four hour intervals under the same conditions. The birth weight of the male twin was 6 pounds and 3 ounces (2.80 Kg.) and that of the female 6 pounds and 4 ounces (2.83 Kg.). The temperatures were normal and neither showed any disturbances throughout this period of observation. The male twin lost 2 per cent of his weight while the female twin lost 5 per cent. The male twin gained progressively immediately after regaining his birth weight on the fourth day, while the female twin did not regain her birth weight until after the fourteenth day (table 6).

The nutritional progress of the new-born under observation depended on the nature of the conditioning to both breast and bottle. Early in the course of these studies it became apparent that the amount of fluid ingested depended on the delicacy of the care and the manner of the administration of feeding. In spite of the intrinsic differences in the new-born infants, it was observed that the more favorable the conditioning to the feeding during the first days of life the greater was the total fluid intake and the less the postnatal loss in weight. Table 9 reveals how markedly the fluid intake may be diminished in untrained hands with improper psychologic conditioning to the feeding procedure.

Maximal fluid intake was realized by gentle handling of the new-born infant. He was never aroused abruptly and never smacked on the heels to be awakened. The head and back were carefully supported when he was lifted from the crib. He was carefully cradled in the nurse's arms with the head slightly elevated and not fed until serene adjustment between the two was obtained. The nipple was carefully washed to eliminate the presence of even minute foreign matter, and it was never thrust into the mouth but rather offered insistently until it was held firmly. The hydrating solution retained its temperature for longer periods of time than either water or the formulas, and was not a negligible influence in the continuity of the feeding. The solution was never forced when it became apparent from gestures of the head that the baby would take no more, thus obviating regurgitation. The baby was then allowed to rest for a few minutes before he was lifted

over the nurse's shoulder for the escape of gas. It was found that rubbing the back was more agreeable than patting. The infant was then replaced in a crib slightly elevated at the head, for elevation of the feet was more prone to produce subsequent regurgitation.

Similar caution was exercised in the manner of nursing. New-born infants fed without supreme regard to their psychologic sensitivity to their surroundings did not respond as favorably so far as feeding effectiveness was concerned. When it is remembered that the psychic mechanism in man determines all other alimentary functions, it is not surprising that the bodily sensitivity of the new-born infant should so alter his alimentary response.

Proper conditioning coupled with the continuous administration of the solution throughout the twenty-four hours resulted in considerable intake in comparison with the controls. New-born infants offered water

TABLE 9.—*Effect of Proper Conditioning on Fluid Intake*

Psychologic Conditioning				Routine Procedure			
Infant	Total Fluid Intake, Oz.	Birth Weight	Average Weight Lost, per Cent	Infant	Total Fluid Intake, Oz.	Birth Weight	Average Weight Lost, per Cent
P	147	7 lbs. 3 oz.	0.8	F	51.5	7 lbs. 2 oz.	9.1
D	124	7 lbs. 5 oz.	0.8	D	60	7 lbs. 13 oz.	6.0
P	136	5 lbs. 4 oz.	0.0	F	68	7 lbs. 7 oz.	5.0
S	87	6 lbs. 10 oz.	0.0	C	59	8 lbs. 3 oz.	5.6
M	129	8 lbs. 1 oz.	3.8	C	66	7 lbs. 15 oz.	5.5
R	117	8 lbs. 12 oz.	3.2	E	54	6 lbs. 10 oz.	5.7
F	119	6 lbs. 6 oz.	2.2	D	68	7 lbs. 7 oz.	5.8
H	186	8 lbs. 2 oz.	0.0	C	126	8 lbs. 6 oz.	1.4
W	174	8 lbs. 6 oz.	0.0	C	112	5 lbs. 14 oz.	3.7
B	180	7 lbs. 14 oz.	0.3	B	110	8 lbs. 12 oz.	2.5

between nursing showed a total volume intake much below physiologic requirement during the first week of life. New-born infants on complemental feeding and nursing ingested larger amounts of total fluid but nowhere near the total intake of those who were offered the hydrating solution at two hour intervals throughout the twenty-four hour cycle (table 9). If we were to abide by the physiologic capacity of the stomach day by day after birth we should be content with the lower curve, indicating the intake on complemental feeding and nursing.

The clinical superiority of the new-born infants who received the hydrating solution led us to favor the latter procedure on the basis of its maximum effectiveness (fig. 4). The considerable fluid intake shown by the new-born infants receiving the hydrating solution is, of course, paralleled by an increased caloric intake in comparison with the controls during the critical first three days of life. It is to be reiterated that the primary purpose was not caloric adequacy but rather fluid adequacy, although it was intended to offer the new-born at least the caloric requirement essential for basal activity, that is, at least 150 calories for the first twenty-four hours (table 10).

New-born infants adequately hydrated rapidly lose the so-called physiologic apathy, somnolence and stupor. New-born infants maintained on water the first three days in addition to the inadequate supply of breast milk not infrequently showed acetonuria. It has already been universally demonstrated that the compensated acidosis of the new-born is an entity which undoubtedly contributes to the familiar apathy of the new-born. The administration of the hydrating solution effectively cleared this needlessly accepted symptomatology.

Adequate hydration of the new-born infant freed him from the familiar starvation stools. On the "solution" the meconium stools were larger and more rapidly eliminated during the first three days of life than in the control series, in which elimination of meconium continued up to the fifth day. Pediatricians have often associated prolonged elimination of meconium stools with periodicity of alimentary disturbances. The toxic symptoms which have been found to supervene have made it a practice among many to resort to castor oil. We have observed

TABLE 10.—*Average Caloric Intake in Treated and Control Series*

Birth Weight, Lbs.	Treated Series			Control Series		
	1 Day	2 Days	3 Days	1 Day	2 Days	3 Days
9	209	235	245	73	116	131
8	142	163	185	86	120	136
7	166	171	225	73	108	120
6	156	188	198	68	116	121
5	163	189	193	..	...	...

that such a procedure aggravated the dehydration even though it cleared the transient disturbances. The new-born infants under observation showed no such upsets and required no special alimentary therapy. Their temperatures were better stabilized in comparison with controls. In fact, the fluctuant variation in temperature in the new-born has led many to attribute it to an immature temperature-regulating mechanism. We have observed the smallest number of febrile infants throughout the year of this study, and none with dehydration fever.

CHANGES IN THE BLOOD OF TREATED NEW-BORN INFANTS

Comparative observations of the clinical course of new-born infants receiving the solution were correlated with studies of the blood. They involved daily microdeterminations in the nursery of the biochemical changes induced during the first fortnight of life as a result of this procedure. Daily determinations of the blood sugar content after fasting and the sugar tolerance curve indicated the new-born infant's need for and response to readily available carbohydrate. Determinations of the refractive index and viscosity revealed the degree of hydration at birth and its subsequent course after administration of the solution.

It further confirmed the hydrophilic state of the plasma proteins. Determination of the clotting and bleeding times daily revealed the relationship between ingested gelatin and blood clotting function.

Blood Sugar.—Determinations of blood sugar after fasting were made daily, by the second micromethod of Folin and Svedberg,[26] in infants receiving the solution as well as in controls. Immediately after birth, we observed uniformly a striking hypoglycemia, the average value being 75 mg. per hundred cubic centimeters. This confirms similar observations made by Schretter,[27] Styrikowitsch,[28] Greenwald [29] and Brown.[30] Comparative studies of the blood sugar after fasting, in new-born infants of various weights, showed no specific correlation. In the control series the blood sugar curve for the first fortnight paralleled rather closely the weight curve. The blood sugar curve fluctuated at low levels, decreasing to the low point of inflection with the weight curve between the third and fifth days. It was this apparent relationship between blood sugar content and body weight that led us all the more to include dextrose in the solution devised for the prevention of loss of weight in the new-born.

The blood sugar rose rapidly to the normal level on about the third day following the administration of the solution. This level was maintained throughout the baby's stay at the nursery for the fortnight. The course of the blood sugar levels of the controls was, as previously mentioned, comparable to the changes in the weight curves. The birth level of blood sugar was maintained for the first few days in some and diminished in others to the low point on about the fourth day, when it rose again, paralleling the ingestion of milk. On comparing the course of the average blood sugar curve for those infants receiving the solution with the controls, there is observed a stability of blood sugar level in the former and a marked lability in the latter group.

Sugar tolerance curves in new-born infants differ from those in older children. The low initial blood sugar level may be the result of an equilibrium with the maternal blood, which is hypoglycemic because of the lactating process. To what extent it is a result of birth shock is under investigation. Lindberg [31] has shown that on fasting the blood sugar decreases slowly in infants because the glycogen stores approach their minimum and the body must rely chiefly on catabolized tissue protein for continuance of the dextrose supply. The early attainment

26. Folin, O., and Svedberg, A. J.: J. Biol. Chem. **88**:85, 1930.
27. Schretter and Nevinny: Ztschr. f. Geburtsh. u. Gynäk. **98**:258, 1930.
28. Styrikowitsch: Ztschr. f. Kinderh. **47**:517, 1929.
29. Greenwald, H. M., and Pennell, S.: The Carbohydrate Metabolism of the Normal New-Born Infant, Am. J. Dis. Child. **39**:281 (Feb.) 1930.
30. Brown, M. J.: Quart. J. Med. **18**:175, 1925.
31. Lindberg, A.: Ztschr. f. Kinderh. **15**:84, 1916.

of this minimum in the new-born is evidence that they tend to utilize and store carbohydrate rapidly as well as to exhaust their endogenous supply more rapidly than older children. Attempts at determinations of blood sugar tolerance in two premature infants revealed an embryonal type of curve, while in other new-born infants the sugar tolerance curve simulated a protein tolerance curve in some and a fat tolerance curve in others. The majority, however, showed a typical curve without attaining the usual peak observed in older children.

The hypoglycemia of the new-born appears to parallel the tendency toward a subnormal temperature. Subnormal temperatures have further been obviated by omission of the initial water bath to prevent chilling, and by cleansing with liquid abolene instead. Apparently trauma to the vegetative nervous system results in cooling, and the subsequent suppression of glycogenolysis is reflected in the hypoglycemia.

Refractive Index.—Daily determinations of the refractive indexes were made by means of the Pulfrich refractometer. A capillary tube about 6 cm. in length was filled with blood to the point *A* as shown in figure 6. In order to prevent the blood from being heated during the stated process, it was drawn up into the tube to 1 cm. from the capillary end. The tube was then sealed in a microburner and inserted in a one-hole rubber stopper and centrifugated for about ten minutes under a drop of oil. A scratch was then made with a file at the point *B*, the boundary line of the serum, and the tube broken at this point. The serum was transferred to the prism of the refractometer and the index of refraction measured at room temperature.

The refractometry of the blood is a simple method of determining water metabolism. The refractive index of the serum depends largely on its protein content, and concentration changes therein are additive refractively. It has been shown that salts have an inappreciable effect on the refractive index even if doubled in concentration in the blood serum. The protein concentration is eight times the salt content and thirteen times that of the organic salts present. Serum protein is therefore the controlling factor, and since it is directly related to the degree of hydration it becomes a measure of the water metabolism. Already, Rott [32] has shown that the refraction curve is a true mirror image of the weight curve in the new-born.

The refractive index of the treated new-born infants remained markedly constant throughout the fortnight of their stay in the nursery. This refractive stability is indicative of invariant hydration in comparison with the controls. They showed an initial diminution in the refractive index (or an increase according to the Pulfrich scale), which

32. Rott: Ztschr. f. Kinderh. **1**:43, 1910.

gradually rose as milk or fluids were offered. Determinations on the serum instead of the whole blood, of course, obviated the gross error of erythrocyte hemolysis, which would be reflected in the refraction determinations.

Viscosity.—Viscosity of the blood is affected by corpuscles in suspension as well as by the proteins according to their degree of hydration. The determinations were made by means of a microviscosimeter provided with a water jacket. The values given for the viscosity of the blood are relative to those for water of the same temperature. We observed a marked initial viscosity of the blood from two to three times that of the norm. This rapidly decreased to about one-half its initial value on the third day and then gradually veered toward the normal, which is 5 in comparison with water as unity. In view of the rapid erythrocyte hemolysis occurring during the first week of life, similar determinations of viscosity were made on the blood serum. Once hydration was maintained by the administration of the solution immediately after birth, the viscosity curve for serum simulated that of the refractive index. The control group, however, showed a prolonged serum viscosity which approached normal values after an adequate intake of fluid was established. Comparison of the viscosity curve with that of the refractometry curve in the control series reveals that dehydration of the new-born with a high viscosity of the blood and a normal refractive index is indicative of a state of shock during the first days of life.

Clotting Function.—New-born infants treated with the solution showed diminished clotting and bleeding time in comparison with the controls. Although these determinations have no direct bearing on blood and tissue hydration in the new-born, we wish to record a favorable effect on the blood clotting function produced by the gelatin. Routine determinations of clotting and bleeding time initiated in previous years aimed to reveal a prolonged clotting time in the new-born on the first day of life for the early diagnosis of potential hemorrhagic disease. As a result we have previously observed in about two hundred new-born infants an average clotting time of about eight minutes; these included several new-born infants with hemorrhagic disease with clotting times ranging up to one hundred and twenty minutes. Curiously enough, throughout the year of study of new-born infants for birth shock and the prevention of initial loss of weight, we have not observed a single infant with hemorrhagic disease.[33]

The average clotting time was reduced for the present series to about three minutes from an initial average of about eight minutes, the lower clotting level assuming constancy after the third day of life. This

33. Kugelmass, I. N.: M. Clin. North America **14**:1523, 1931; **15**:1313, 1932.

stability of the clotting mechanism was not observed in the control series. Similarly, the determination of bleeding time on the lobe of the ear remained at the average low level of a little under a minute after the second day of life in comparison with markedly fluctuant bleeding time values for the control series. Blood clotting function can never be evaluated in terms of clotting and bleeding time except in the new-born. Cumulative environmental changes affect the clotting mechanism to no small degree. As Sanford [34] has recently confirmed, the new-born can safely be studied, therefore, from this standpoint by simple clinical clotting and bleeding time values as criteria of their tendency to bleed.

One of us (Dr. Kugelmass [35]) has already demonstrated in a series of studies of the relation between dietary factors and blood clotting function that protein accelerates the tendency to clot. Similarly, by a dietary excessively high in protein, he was able to prevent hemorrhagic disease of the new-born in the offspring of a woman who had previously given birth to four babies with the disease.[36] The mother's blood showed a low prothrombin content, which was subsequently found to be characteristic of many new-born infants with hemorrhagic disease. Further work has definitely indicated that high protein ingestion increases the prothrombin content of the blood. Therefore it is reasonable to assume that in the present series of new-born infants given the special solution that the gelatin was operative in accelerating the clotting mechanism to the extent that it became manifest in the markedly low clotting time. It is of advantage to new-born infants to have a diminished tendency to bleed, and the ingestion of gelatin brings about this tendency.

The consistently low bleeding time values cannot be correlated with the effect of gelatin because it is more directly related to the platelet content, which is normal but variant in new-born infants. Our previous studies, however, have indicated that the administration of gelatin condenses platelets on the vascular bed and so indirectly reduces the bleeding time. The whole effect of the ingestion of gelatin during the first days of life has been to stabilize the blood clotting function in comparison with that of the control series, which revealed markedly fluctuant values day by day.

---

34. Sanford, H. N.; Gasteye, T. H., and Wyat, L.: The Substances Involved in the Coagulation of the Blood of the New-Born, Am. J. Dis. Child. **43**:18 (Jan.) 1932.

35. Kugelmass, I. N., and Samuel, E. L.: Dietary Protein and Blood-Clotting Function, Am. J. Dis. Child. **41**:48 (Jan.) 1931.

36. Kugelmass, I. N., and Tritsch, J. E.: Prenatal Prevention of Potential Hemorrhagic Disease of the New-Born, J. A. M. A. **92**:531 (Feb. 16) 1929.

CONCLUSIONS

1. Loss of weight in the new-born is sanctioned universally without physiologic foundation.

2. The new-born infant is markedly underdeveloped for the duration of human gestation, and so requires continuous supervision for the prevention of potential pathologic changes.

3. The neonatal growth gradient continues unaltered during the postnatal period according to analysis of the transitional growth trends of the body as a whole as well as of its tissues and organs; therefore the initial loss in weight is necessarily extrinsic.

4. Postnatal loss of weight does not obtain in animals of any size, gestation period or scale in evolution, according to a survey of observations of veterinarians, animal husbandmen and curators in experimental and zoological parks in this country and abroad.

5. The initial loss of weight in the new-born is the result of dehydration and semistarvation, conditions unfavorable for nutritional, physical and environmental adjustments besetting the new-born.

6. Past therapeutic procedures for decreasing the loss of weight in the new-born have not been altogether effective because they were not based on the physiologic needs of the new-born disturbed by birth shock.

7. The initial loss in weight in the new-born can be prevented by the administration of a solution consisting of 6 per cent gelatin (p_H 6.2), 3 per cent dextrose and 0.5 per cent sodium chloride at two hour intervals throughout the twenty-four hour cycle immediately after birth. The gelatin hydrates blood and tissues; it raises body heat by virtue of its specific dynamic action, and it reduces the clotting time. Dextrose brings the hypoglycemia of the new-born to normal. Sodium chloride raises the initial low blood chloride and favors hydration.

8. The average loss of weight in new-born infants receiving the hydrating solution was 1.7 per cent, the irreducible minimum in comparison with the average loss of 7 per cent in the controls.

9. The characteristic clinical picture of the new-born is a result of birth shock, more effectively combated by the hydrating solution than by milk mixtures the first two or three days of life.

10. The total fluid intake of new-born infants properly conditioned to both breast and bottle was as much as twice that of the series receiving routine nursery care.

11. Preventing loss of weight in the new-born produces rapid disappearance of the so-called physiologic apathy, somnolence and stupor secondary to birth shock and the compensated acidosis universally present.

12. Intrinsic differences in the new-born infants studied revealed that male infants lost more than female infants, that first-born infants tended to lose more than siblings, that babies in the ward lost more than babies in private cases, and that those of Latin extraction lost more than Nordics.

13. The clinical course of the new-born infants was correlated with daily studies of the blood sugar and sugar tolerance curve, the refractive index and viscosity, the acid-base equilibrium and the clotting and bleeding times during the first fortnight of life.

14. New-born infants show a hypoglycemia the first days of life and a sugar tolerance curve of low peak, thus indicating a dire need for carbohydrate as well as a tendency to utilize, store and exhaust the endogenous supply of carbohydrate more rapidly than older children.

15. Determinations of the refractive index and viscosity on the serum of new-born infants revealed concentration of the blood on the first days of life, gradually attaining normal values following the administration of food in the control series; however, the new-born infants treated with the hydrating solution showed markedly constant refractive and viscosimetric curves throughout.

16. The gelatin component of the hydrating solution decreased the clotting time to less than three minutes in comparison with seven minutes for the control series.

CARBOHYDRATE METABOLISM

I. EFFECT OF PREVIOUS DIET ON UTILIZATION OF GLUCOSE INJECTED INTRAVENOUSLY

J. A. JOHNSTON, M.D.
DETROIT

In the course of some observations made five years ago on children recovering from severe bouts of vomiting, I noted a failure of elevation of the respiratory quotient following the intravenous administration of glucose. Because such states involve so many variables, viz., an accompanying infection, emotion, alteration in the acid-base balance or similar influences, the present studies were undertaken in order to analyze further the factors affecting the utilization of intravenously administered glucose.

Lehmann [1] in 1874 and Hofmeister [2] in 1890 described "diabetes" due in the first instance to either arsenic poisoning or starvation and in the second to starvation. The similarity of the states induced by arsenic poisoning and by starvation naturally suggested that the site of the metabolic flaw was in the liver. This idea was further developed by the work of Barrenschen [3] in 1914 and by that of Nagasuye [4] in 1925. The reproduction of the "high prolonged" so-called "diabetic" sugar tolerance curve as a result either of starvation or of diets that might be expected to deplete the supply of reserve carbohydrate has been accomplished by a great many workers. Staub [5] produced this effect after two days of a protein-fat diet but obtained no effect after a single meal. He concluded also that a fast of longer than fifteen hours produced a diabetic curve. When work was done immediately after the tolerance test he found an acceleration of the removal of sugar from the blood,

---

Read before the Pediatric Research Society, Atlantic City, N. J., May, 1932.

From the Department of Pediatrics and the Department of Laboratories, Henry Ford Hospital.

1. Lehmann, W. L.: Het arsenigzuur als geneesmiddel by diabetes mellitus, Amsterdam, C. G. van der Port, 1873; quoted by Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1931, vol. 1, p. 122.

2. Hofmeister, F.: Ueber Resorption und Assimilation der Nahrstoffe: VI. Ueber den Hungerdiabetes, Arch. f. exper. Path. u. Pharmakol. **26**:355, 1889-1890.

3. Barrenschen, H. K.: Ueber Glykogen und Zuckerbildung in der isolierten Warmbluterleber, Biochem. Ztschr. **58**:277, 1913.

4. Nagasuye, S.: Experimental Investigation on Assimilation of Levulose, Galactose and Dextrose During Fasting and with Albumin-Fat Diet, Biochem. Ztschr. **5**:449, 1925.

5. Staub, H.: Untersuchungen über den Zuckerstoffwechsel des Menschen, Ztschr. f. klin. Med. **93**:89, 1922.

but if the work preceded the giving of glucose he found a high prolonged curve (evidence of the effect of depleted stores). It is worth noting that the altered curve produced by Southworth [6] could be rendered normal by the administration of insulin. Sweeney's [7] best curves were obtained after a carbohydrate feeding, and his highest (i. e., most defective) curve after fasting and the administration of diets high in fat. Gilchrist [8] felt that the alteration following ketosis was the effect of depleted stores rather than of the change in acidity.

Increased utilization as measured by tolerance curves was shown by Lennox [9] to follow both the ingestion and the injection of glucose.

The little work that is available to show that the phenomenon involves a diminution in actual oxidation was reviewed by Dann and Chambers,[10] who demonstrated in fasting dogs the failure of the elevation of the respiratory quotient following the injection of glucose. There should be added to their account, however, the work of Bernstein and Falta [11] in 1918, who demonstrated a failure of elevation of the respiratory quotient following oral injection of carbohydrate when the glycogen stores were depleted. Curiously, they claimed "after intravenous sugar a distinct rise in the R. Q. irrespective of whether the glycogen stores are perfectly filled."

METHODS

The method was similar to that employed by Mason [12] and by Linder, Hiller and Van Slyke,[13] although the latter drew their conclusions from curves of the nonprotein respiratory quotient. The subjects were well trained and were old enough to cooperate perfectly. The fasting metabolism was determined under basal conditions; blood was taken for determination of the p_H, bicarbonate and sugar; 40 cc. of 50 per cent glucose was then injected very slowly, and additional determinations of metabolism were made for the last eight to ten minutes of the thirty, sixty, hundred and twenty and hundred and eighty minute periods. The subject voided about two hours before the first test; urine was then collected at inter-

6. Southworth, A. R.: Effects of Carbohydrate Deprivation upon Pancreatic Function, M. J. Australia **2**:460, 1923.

7. Sweeney, J. S.: Dietary Factors that Influence the Dextrose Tolerance Test; Preliminary Study, Arch. Int. Med. **40**:818 (Dec.) 1927.

8. Gilchrist, M. L.: The Influence of Acidosis on Carbohydrate Metabolism, Arch. Dis. Childhood **7**:169, 1932.

9. Lennox, W. G.: Repeated Blood Sugar Curves in Non-Diabetic Subjects, J. Clin. Investigation **4**:331, 1927.

10. Dann, M., and Chambers, W. H.: Animal Calorimetry: Metabolism of Glucose Administered to Fasting Dog, J. Biol. Chem. **89**:675, 1930.

11. Bernstein, S., and Falta, W.: Respiratorischer Stoffwechsel und Blutzuckerregulation, Deutsches Arch. f. klin. Med. **125**:233, 1918.

12. Mason, E. H.; Hill, E., and Charlton, D.: Abnormal Specific Dynamic Action of Protein, Glucose, and Fat Associated with Undernutrition, J. Clin. Investigation **4**:353, 1927.

13. Linder, G. C.; Hiller, A., and Van Slyke, D. D.: Carbohydrate Metabolism in Nephritis, J. Clin. Investigation **1**:247, 1925.

vals for a period of about six hours, ending two hours after the last test, and was analyzed for nitrogen and sugar. Calculation of the metabolic mixture was made from the table of Lusk.[14] When disturbance of acid-base balance might be anticipated, bicarbonate was estimated at the beginning and end of the morning, and correction was made for changes according to the method described by Peters and Van Slyke [15] and based on the data of Shaw.[16] The determinations of metabolism were carried out by the gasometer method. Gas analyses were made in duplicate or triplicate with the Haldane-Henderson apparatus, samples of approximately 9.5 cc. being used. Blood sugar was determined by the method of Folin and Wu.[17] Blood for determinations of the p_H and bicarbonate content was drawn under oil and delivered under oil into special centrifuge tubes, according to the technic described by Myers.[18] Determinations of p_H were made colorimetrically by Myers and Muntwyler's [19] adaptation of the Cullen method. For determinations of the bicarbonate content, samples of plasma were withdrawn from under the oil and analyzed without reequilibration, by the method of Van Slyke and Stadie.[20] Urine was analyzed for nitrogen by the Kjeldahl-Gunning [21] procedure and for sugar by the method of Benedict.[22]

Certain limitations of the method prevent an exact quantitative treatment of the results. The calculation of an hourly value from an eight or ten minute measurement is, of course, open to objection, although a fairly constantly dropping curve in the second hour will roughly balance an increasingly rising one in the first hour. However, a longer period even when a comfortably fitting mask is used causes restlessness. The partitioning of the nitrogen, as Linder and his associates pointed out, involves a hopeless difficulty because of the nitrogen lag. In the hour immediately following the administration of glucose, even though the respiratory quotient rises abruptly, there is usually a sharp rise in nitrogen, presumably due to a sweeping out. However, merely to average the results for the entire six hour period would neglect an occasional striking fall of nitrogen in the third hour due to the sparing effect of carbohydrate. My associates and I have tried to average or partition, depending on whether or not the differences were great. The injection of so strong a solution of glucose, unless it is done very slowly, always results in some glycosuria. In about one hundred experiments we have found an average loss of sugar under normal conditions of about 1.25 Gm. The use of a more dilute solution for injection involves the variable of hydration, which is one item that we are trying to avoid. Comparative conclusions are drawn from a consideration of

14. Lusk, G.: Animal Calorimetry; Analysis of Oxidation of Mixtures of Carbohydrate and Fat, J. Biol. Chem. **58**:41, 1924.

15. Peters, J. P., and Van Slyke, D. D.: Quantitative Chemistry, Baltimore, Williams & Wilkins Company, 1931, vol. 1, p. 17.

16. Shaw, L. A.: Comparative Capacity of Blood and of Tissue to Absorb Carbonic Acid, Am. J. Physiol. **79**:91, 1926.

17. Folin, O., and Wu, H.: System of Blood Analysis; Simplified and Improved Method for Determination of Sugar, J. Biol. Chem. **41**:367, 1920.

18. Myers, V. C.: Practical Chemical Analysis of Blood, ed. 2, St. Louis, C. V. Mosby Company, 1924.

19. Myers, V. C., and Muntwyler, E.: Colorimetric Estimation of Hydrogen Ion Concentration of Blood, J. Biol. Chem. **78**:243, 1928.

20. Van Slyke, D. D., and Stadie, W. C.: Determination of Gases of Blood, J. Biol. Chem. **49**:1, 1921.

21. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1932, vol. 2, p. 56.

22. Benedict, S. R.: The Detection and Estimation of Glucose in Urine, J. A. M. A. **57**:1193 (Oct. 7) 1911.

TABLE 1.—*Effect of Starvation on M. K., an Epileptic Patient*

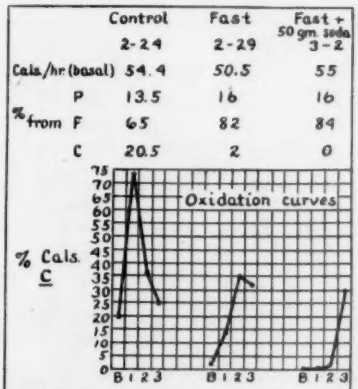
Date	Weight, Pounds	Estimated Weight, Pounds	Diet: Calories	Diet: Per Cent Calories: Protein	Diet: Per Cent Calories: Fat	Diet: Per Cent Calories: Carbohydrate	Metabolism	Calories	Gm.: Protein	Gm.: Fat	Gm.: Carbohydrate	Glycosuria	Basal Available Glucose†	Gm. Carbohydrate Oxidized in 4 Hours	Ratio
2/24	103	110	1,746	14	66	20	B*	54.43	1.79	3.86	2.73	0	4.25	18.99	1:4.4
							30	27.69	0.89	1.16	3.22	Trace			
							60	27.70	0.89	0.38	5.00	1.08			
							120	52.58	1.79	2.82	4.63	Trace			
							180	52.94	1.79	3.00	3.41	0			
2/29	100	...	50 Gm. lettuce				B	50.56	1.95	4.43	0.30	0	1.87	8.80	1:4.7
			50 Gm. celery				30	25.89	0.97	2.11	0.56	1.23			
			Water ad libitum				60	24.01	0.97	1.61	1.23	0			
							120	48.82	1.95	2.55	4.18	0			
							180	47.29	1.95	2.65	2.58	0			
3/ 2	98	...	50 Gm. lettuce				B	55.41	2.18	5.00	0.00	0	1.26	4.88	1:3.3
			50 Gm. celery				30	21.59	1.09	1.70	0.32	1.58			
			50 Gm. sodium bicarbonate				60	21.03	1.09	1.78	0.00	0			
			Water ad libitum				120	48.90	2.18	4.29	0.18	0			
							180	50.58	2.18	2.78	3.85	0			

* In this column, B is the basal hour; the figures below represent minutes after the intravenous injection of 20 Gm. of glucose.
† Derived from 6.58 Gm. protein plus 0.1 Gm. fat plus carbohydrate of the basally oxidized mixtures.

data derived from alterations in variables in the same subject, and accordingly the amount of glucose was kept constant in all of the experiments rather than a different dose by weight estimated for each patient.

EFFECT OF STARVATION

CASE 1.—M. K., a girl, aged 17, was observed because of epileptic seizures from Dec. 28, 1931, to Feb. 29, 1932. The treatment consisted of the administration of ketogenic diets of increasing strength from protein 54 Gm., fat 113 Gm. and carbohydrate 92 Gm. to protein 60 Gm., fat 205 Gm. and carbohydrate 85 Gm. With these diets the weight remained at 103½ pounds (46.9 Kg.). The first control determination was made while the patient was still on the last diet. Following this a period of almost complete starvation was instituted, the patient being allowed by mouth only 50 Gm. of lettuce and 50 Gm. of celery daily. Water was allowed



	Control	Fast	Fast + 50 gm. soda
	2-24	2-29	3-2
Cals./hr. (basal)	54.4	50.5	55
% from P	13.5	16	16
% from F	65	82	84
% from C	20.5	2	0
Initial pH	7.46	7.38	7.38
" CO_2	58	41.6	59.3
Blood sugar	76	57	56
Final pH	7.45	7.40	7.42
CO_2	58	49.4	57.2
Gm. C oxidized	18.9	8.8	4.2
Urine sugar	1.08	1.23	1.58

Chart 1.—Data for M. K., showing the effect of fasting on (*a*) the basal metabolic mixture, (*b*) the curve of oxidation of administered glucose, (*c*) the fasting blood sugar and (*d*) changes in p_H and bicarbonate.

and was freely taken (up to nearly 6,000 cc. on the third day of the fast). Further observations were made on the fifth and seventh days of the fast. The data are recorded in table 1. On the day preceding the final determination, 50 Gm. of sodium bicarbonate was given. Discussion of the possible effect of this particular part of the experiment is deferred to another communication which will consider the possible relationship of the p_H to the glucose oxidized.

The basal mixture in the control determination showed a lower percentage of carbohydrate than is usually found, but this is readily understood as a result of the ketogenic diet. The curve of oxidation of the administered glucose was normal (chart 1). Fasting resulted in a fall in total calories, a rise in the percentage of calories derived from protein and fat and a rapid diminution in the percentage derived from carbohydrate. The carbon dioxide fell from 58 to 49.4 cc. per hundred centimeters of plasma on the fifth day; it rose to 59.3 cc. following the administra-

tion of sodium bicarbonate without a coincidental rise in p_H. The weight dropped 3 pounds (1.4 Kg.) in five days and 5 pounds (2.3 Kg.) in seven days. This loss was accompanied by a profound diuresis, the volume of urine exceeding the volume of a very large voluntary intake of water. The curve of oxidation of administered glucose after five days reached its peak at the end of the second hour, and after seven days the peak was reached at the end of the third hour. The number of grams of carbohydrate oxidized was 8.8 after five days as compared with the control value of 18.9 and fell to 4.2 on the seventh day. The amount lost in the urine showed a negligibly small increase, certainly nothing that would enter into the explanation of the disappearance of the sugar not oxidized.

EFFECT OF CHANGE OF DIET

In tables 2, 3 and 4 are given the detailed data on three normal girls. A. K., aged 12, was a slender, undernourished child, 9 pounds

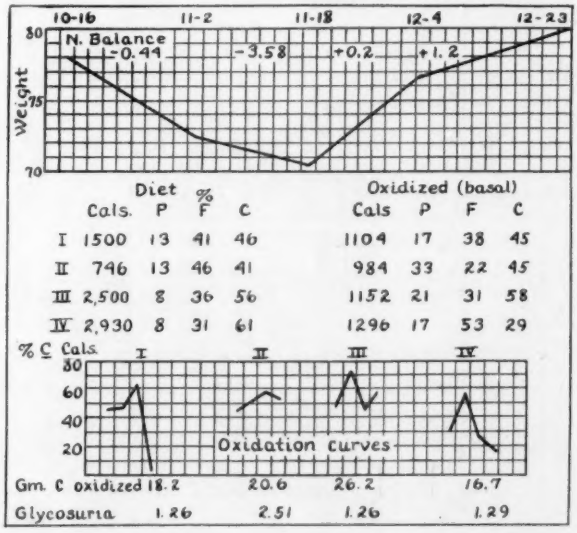


Chart 2.—Composite graph for A. K., showing diets and the composition of the oxidized mixtures.

(4.1 Kg.) under the estimated weight. G. K., aged 13, was overweight (122 pounds [55.3 Kg.]; estimated weight, 97 pounds [44 Kg.]). H. M., aged 13, appeared on examination to be definitely malnourished; she had a positive tuberculin test and a calcified node in the hilus, but no evidence of an active disease process. All three subjects were given measured diets over a period of four months.

The essential points to be noted are that, as might be expected, the submaintenance diet results in a fall in total calories, a rise in protein and fat and a fall in carbohydrate. The last, however, did not occur in A. K., the undernourished child, on her lowest diet; the number of calories fell, and the protein was doubled, but fat and carbohydrate remained about the same. The essential points in this case are shown more graphically in chart 2. The nitrogen balance indicated here was

TABLE 2.—*A. K. (Normal with Diet Variation)*

Date	Weight, Pounds	Estimated Weight, Pounds	Diet				Metabolism						Basal Available Glucose†	Gm. Carbohydrate Oxidized in 4 Hours	Ratio
			Calories	Per Cent Calories				Calories	Gm.			Glycosuria			
				Protein	Fat	Carbohydrate			Protein	Fat	Carbohydrate				
11/ 3	70¼	79	1,500	13	46	41	B*	46.33	1.91	1.90	5.08	0	6.3	18.24	1:2.9
							60	48.66	2.04	1.84	5.65	1.26			
							120	48.00	2.41	0.79	7.51	0			
							180	47.25	1.84	4.27	0.00	0			
11/18	70½	..	746	13	46	31	B	41.46	3.32	0.97	4.59	0	6.61	20.67	1:3.1
							60	40.11	3.35	0.64	4.98	2.51			
							120	40.40	2.77	0.60	5.71	0			
							180	40.67	2.30	0.98	5.39	0			
12/ 3	76	..	2,500	8	36	56	B	48.21	2.57	1.59	5.59	0	7.24	26.16	1:3.6
							60	48.38	2.56	0.26	8.65	1.26			
							120	48.31	2.81	1.73	5.04	0			
							180	47.39	2.28	1.06	6.88	0			
12/23	77½	..	2,900	8	31	61	B	54.30	2.24	3.13	3.91	0	5.52	16.76	1:3.0
							60	52.87	2.51	1.42	7.16	1.29			
							120	50.94	2.65	2.80	3.41	0			
							180	51.97	2.65	3.41	2.28	0			
1/28	82	..	2,000	17	37	46	B	54.37	2.90	2.88	3.82	0	5.78	15.83	1:2.74
							60	50.34	2.90	2.13	4.55	1.54			
							120	51.44	1.96	2.85	4.08	0			
							180	52.89	1.96	3.23	3.38	0			

* In this column, B is the basal hour; the figures below represent minutes after the intravenous injection of 20 Gm. of glucose.
† Derived from 0.58 Gm. protein plus 0.1 Gm. fat plus carbohydrate of the basally oxidized mixtures.

TABLE 3.—*G. K. (Normal with Diet Variation)*

Date	Weight, Pounds	Estimated Weight, Pounds	Diet: Calories	Diet: Per Cent Calories: Protein	Diet: Per Cent Calories: Fat	Diet: Per Cent Calories: Carbohydrate	Metabolism	Calories	Gm.: Protein	Gm.: Fat	Gm.: Carbohydrate	Glycosuria	Basal Available Glucose†	Gm. Carbohydrate Oxidized in 4 Hours	Ratio	Comment
10/28	116	97	2,000	17	37	46	B*	57.86	2.38	3.44	3.92	0	5.64	14.32	1:2.5	
							60	58.96	2.44	2.60	6.06	0.45				
							120	60.10	2.44	4.74	1.47	0				
							180	61.77	2.48	4.28	2.87	0				
11/30	118	..	2,300	13	62	25	B	56.72	2.85	3.40	3.29	0	5.28	17.14	1:3.2	Acetone, slight trace; no diacetic acid; p_H 7.57; carbon dioxide 58 mg.
							60	56.35	3.15	2.14	5.73	1.05				
							120	56.85	2.39	2.84	5.03	0				
							180	54.08	2.85	3.20	3.09	0				
1932 2/ 1	120¼	..	2,000	17	37	46	B	56.83	2.93	2.44	5.39	0	7.3	22.14	1:3.0	
							60	54.35	1.56	2.09	6.95	0				
							120	53.58	2.11	2.55	5.18	0				
							180	53.46	1.65	2.90	4.62	0				

* In this column, B is the basal hour; the figures below represent minutes after the intravenous injection of 20 Gm. of glucose.
† Derived from 0.58 Gm. protein plus 0.1 Gm. fat plus carbohydrate of the basally oxidized mixtures.

TABLE 4.—*H. M. (Normal)*

Date	Weight, Pounds	Estimated Weight, Pounds	Diet: Calories	Diet: Per Cent Calories: Protein	Diet: Per Cent Calories: Fat	Diet: Per Cent Calories: Carbohydrate	Metabolism	Calories	Gm.: Protein	Gm.: Fat	Gm.: Carbohydrate	Glycosuria	Basal Available Glucose†	Gm. Carbohydrate Oxidized in 4 Hours	Ratio	Comment
4/14	80¾	88	2,000	17	37	46	B*	47.93	1.32	2.10	5.61	0	6.58	23.74	1:3.5	
							60	46.39	1.32	1.64	6.28	1.89				
							120	47.81	0.07	2.47	5.89	0				
							180	47.38	0.07	2.40	5.96	0				
4/28	75½	..	2,000	..	..	..	B	44.73	2.20	2.88	1.69	0	3.25	7.99	1:2.1	Tonsils removed on April 19; weight decreased 5 pounds
							60	43.27	2.20	7.91	3.38	1.17				
							120	43.33	2.20	2.78	1.43	0				
							180	46.02	2.20	3.04	1.49	0				
6/ 3	83	..	2,800	15	35	50	B	45.98	2.96	1.91	3.92	0	5.82	17.66	1:3.0	
							60	46.74	2.96	1.21	5.70	2.02				
							120	45.95	2.00	2.06	4.54	0				
							180	46.65	2.00	2.58	3.52	0				

* In this column, B is the basal hour; the figures below represent minutes after the intravenous injection of 20 Gm. of glucose.
† Derived from 0.58 Gm. protein plus 0.1 Gm. fat plus carbohydrate of the basally oxidized mixtures.

obtained over periods of several days with the various diets. Aside from the changes when the subjects were on submaintenance diets, the basal metabolic mixture would seem to be relatively independent, within wide limits, of variations in the percentage comparison of the intake.

It is difficult to draw conclusions from differences in the curves of oxidation, save that the curve for the lowest diet is definitely flatter than the rest. When, however, the number of grams oxidized in the entire period is related to the basal metabolic mixture, a definite correlation appears, which will be discussed later.

EFFECT OF HIGH DIET AND REPEATED INTRAVENOUS INJECTIONS OF GLUCOSE

Although the child was not normal, the case of I. P. is included in this presentation because the boy represented a subject for study who for therapeutic reasons was being fed a very high caloric diet and was also being given repeated injections of a 10 per cent solution of glucose.

CASE 2.—I. P., a Jewish boy, aged 12, had suffered from chronic arthitis for four years. In three different hospitals all the conventional therapeutic procedures (removal of foci of infection, vaccine, nonspecific therapy and other measures) had been instituted without beneficial result. Definite improvement both in the patient's general condition and in the spasticity about the joints followed the use of injections of glucose. Part of the interest in studying him arose from an attempt to ascertain whether or not the impaired glucose tolerance reported in chronic arthritis by Pemberton and Foster [23] and by Fletcher [24] was merely the diminution associated with any depletion of body stores resulting from any chronic infection when not offset by a high diet, and to test further whether this could be made to disappear with a filling of stores. The results seem to indicate that there was no specific defect in the oxidation of carbohydrate in this patient with chronic arthritis. In the last three determinations the identical situation was found that obtains in normal persons at various levels of nutrition, there being a direct parallelism between available glucose and oxidized glucose in the first control determination.

COMMENT

In an attempt at a correlation of results with the various factors involved, curves were constructed for A. K. and G. K. as shown in chart 3, and it appears from these that a striking correlation obtains between what we have designated as "basal available glucose" (0.58 Gm. protein + 0.1 Gm. fat + carbohydrate [Woodyatt [25]]) calculated

* In this column, B is the basal hour; the figures below represent minutes after the intravenous injection of 20 Gm. of glucose.
† Derived from 0.58 Gm. protein plus 0.1 Gm. fat plus carbohydrate of the basally oxidized mixtures.

23. Pemberton, R., and Foster, G. L.: Studies on Arthritis in Army Based on 400 Cases; Studies on Nitrogen, Urea, Carbon Dioxid Combining Power, Calcium, Total Fat and Cholesterol of Fasting Blood, Renal Function, Blood Sugar and Sugar Tolerance, Arch. Int. Med. **25**:243 (March) 1920.

24. Fletcher, A. A.: Dietetic Treatment of Chronic Arthritis and Its Relationship to Sugar Tolerance, Arch. Int. Med. **30**:106 (July) 1922.

25. Woodyatt, R. T.: Objects and Method of Diet Adjustment in Diabetes, Arch. Int. Med. **28**:125 (Aug.) 1921.

TABLE 5.—*I. P. (Chronic Arthritic Patient with High Carbohydrate Feeding)*

Date	Weight, Pounds	Estimated Weight, Pounds	Diet: Calories	Diet: Per Cent Calories: Protein	Diet: Per Cent Calories: Fat	Diet: Per Cent Calories: Carbohydrate	Metabolism	Metabolism: Calories	Metabolism: Gm.: Protein	Metabolism: Gm.: Fat	Metabolism: Gm.: Carbohydrate	Glycosuria	Basal Available Glucose†	Gm. Carbohydrate Oxidized in 4 Hours	Ratio	Comment
11/24	53	68	1,500	17	37	46	B*	48.58	1.44	4.00	1.14	0	2.38	10.00	1:4.2	
							60	48.48	1.44	3.00	3.59	0.77				
							120	48.35	1.44	3.15	3.21	0				
							180	52.43	0.67	4.43	2.06	0				
5/12	59¾	..	2,300	15	50	35	B	53.70	1.83	3.80	2.63	0	4.07	11.57	1:2.8	Fatty-acid : glucose ratio of diet = 0.6
							60	50.87	1.83	3.00	3.76	1.82				
							120	49.58	1.83	3.61	2.07	Trace				
							180	51.99	1.83	3.41	3.11	0				
5/27	62½	..	2,300	15	50	35	B	40.90	1.34	1.77	4.63	..	5.58	21.36	1:3.8	
			Plus 1,500 cc. orange juice				60	41.79	1.34	1.11	6.31					
			with 10% lactose + 300 cc.				120	43.67	1.34	1.41	6.12					
			20% glucose daily on				180	43.17	1.34	2.16	4.30					
			May 24, 25 and 26													
5/31	63¾	..	Orange juice, 3,000 cc.				B	53.44	2.93	0.63	8.68	0	10.44	36.43	1:3.5	
			daily + 300 cc. 20% glucose				60	54.97	2.93	0.49	9.37	0.68				
			on May 28				120	53.17	2.93	0.67	8.51	0				
							180	52.88	1.31	0.76	9.87	0				

* In this column, B is the basal hour; the figures below represent minutes after the intravenous injection of 20 Gm. of glucose.
† Derived from 0.58 Gm. protein plus 0.1 Gm. fat plus carbohydrate of the basally oxidized mixtures.

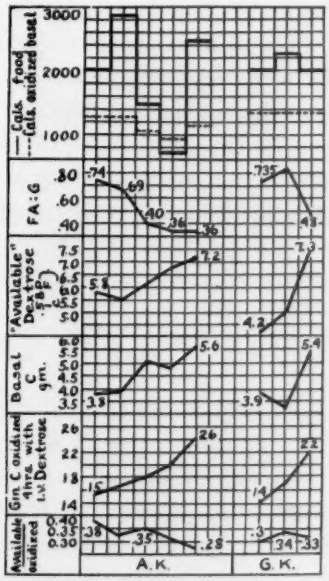


Chart 3.—Simultaneous curves of a number of factors, showing that the best correlation obtains between basal available glucose and oxidized carbohydrate.

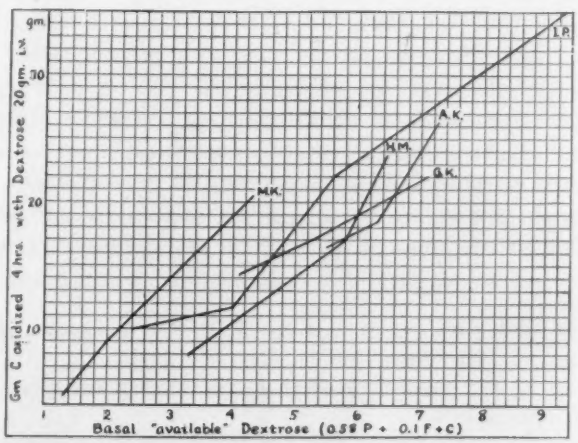


Chart 4.—Composite graph, showing all determinations on the five patients, demonstrating that the relation between basal available glucose and the grams of carbohydrate oxidized after intravenous injection of glucose approaches a straight line.

from the basal metabolic mixture and the number of grams of carbohydrate oxidized in the entire four hours. While it would be simpler to relate the number of grams of sugar oxidized to the number of grams of carbohydrate being oxidized in the basal metabolic mixture, this correlation fails in submaintenance states, only to be compensated for, at least in part, by the rise in protein. A more graphic representation of the results appears in chart 4, in which are plotted simultaneous curves for the five subjects studied; in this chart it appears that the relationship between the basal available glucose and the amount of glucose oxidized following injection is represented by a straight line; as the basal mixture approaches starvation, the ratio of oxidized to available glucose drops until a relative as well as an absolute diminution in oxidation is reached.

That such a situation might obtain (i. e., that protein might increase to compensate for a fall in carbohydrate) was suggested by Gamble, Ross and Tisdall.[26] The rôle of protein as a source of carbohydrate has been particularly stressed by Janney.[27] Cramer's [28] statement of his belief is as follows: "The specific protein-sparing action of carbohydrates in the general metabolism is due to carbohydrate protecting protein from being used by the liver for the formation of glycogen." Markowitz [29] concluded that the formation of glycogen is an obligatory and not an optional step in the formation of blood sugar from the *non-carbohydrate* stores of the organism. In this connection it is worth noting also that du Vigneaud and Karr [30] obtained a better restoration of normal curves by breaking the fast with protein rather than with glucose. All of this involves, of course, the subject of whether or not formation of glycogen is an obligatory step, and while Mann's [31] work throws some doubt on this, it would nevertheless seem to be true that in the general economy only glycogen derived from the liver is utilized. These data do not suffice to furnish an answer to this question but strongly suggest the presence of a certain storage threshold which must be reached before oxidation of administered glucose takes place, a

26. Gamble, J. L.; Ross, G. S., and Tisdall, F. F.: Metabolism of Fixed Base During Fasting, J. Biol. Chem. **57**:633, 1923.

27. Janney, N. W.: Glucose Formation from Protein in Diabetes, Arch. Int. Med. **18**:584 (Nov.) 1916.

28. Cramer, W.: Glycogenic Function of Liver and Its Endocrine Control, Brit. J. Exper. Path. **5**:128, 1924.

29. Markowitz, J.: Glyconeogenesis, Am. J. Physiol. **74**:22, 1925.

30. du Vigneaud, V., and Karr, W. G.: Carbohydrate Utilization: Rate of Disappearance of D-Glucose from Blood, J. Biol. Chem. **66**:281, 1925.

31. Mann, F. C.: Effects of Complete and Partial Removal of Liver, Medicine **6**:419, 1927.

normal physiologic phenomenon which would account in a simple manner for a large number of observations on "diminished tolerance" to carbohydrate.

CONCLUSIONS

1. The amount of intravenously injected glucose oxidized bears a direct relationship to the amount of glucose available in the basal oxidized mixture.

2. The fall in calories and the rise in protein resulting from submaintenance diets and starvation states seem to be a protective mechanism to maintain the level of available glucose.

Dr. O. H. Gaebler made the determinations of p_H and carbon dioxide; Dr. Frank W. Hartman supervised the determinations of metabolism, and Dr. R. J. Mason prepared the patients.

ATMOSPHERIC CONDITIONS IN THE SMALL CANVAS TENT (GUEDEL)

GRACE LUBIN, PH.D.
Chemist, Littauer Pneumonia Research Fund of New York University
AND
JESSE G. M. BULLOWA, M.D.
Clinical Professor of Medicine, New York University
NEW YORK

What are the advantages and shortcomings of the simplest form of oxygen tent, a small canvas hood fitting over the patient's head, supplied with oxygen through an opening at the top? Tents of this type, without motors or cooling units, are in use, especially for children and infants.[1] They can be made at a low cost, are extremely easy to transport and adjust, and, if satisfactory, could certainly be universally available for clinical emergencies. Does this equipment provide a sufficient degree of atmospheric control to warrant continuance of its use, and if so, for what purpose and under what circumstances?

In the present studies, a tent of this type,[2] which was put at our disposal through the courtesy of Dr. Béla Schick of Mount Sinai Hospital, was operated under various conditions, and a record made of the oxygen and carbon dioxide concentrations, the dry bulb temperature, the relative humidity and the movement of air.

In order to evaluate the effect of the tent on the comfort and well-being of the patient as influenced by the rate of heat loss, the last three measurements were combined to give estimates of the effective temperature, calculated according to both the normal and the basic scales of effective or physiologically equivalent conditions as formulated by Houghton, Yaglou and Miller under the auspices of the American Society of Heating and Ventilating Engineers.

In this connection it should be pointed out that the "normal" and "basic" scales of the ventilating engineers are here applied clinically merely for lack of a true clinical scale of effective temperatures for patients in bed, with or without fever. It is to be presumed that either one of the effective temperature scales at present available is a truer guide as to the effect of the environment on the patient than the dry

---

From the Medical Service, Harlem Hospital, Department of Hospitals, and the Littauer Pneumonia Research Fund of New York University.

1. Guedel, Arthur E.: Oxygen Therapy in Pneumonia, J. A. M. A. **84**:1490 (May 16) 1925.

2. Manufactured by the Foregger Company, New York.

bulb temperature alone without consideration of humidity and movement of air. Katathermometer readings during operation would have been desirable, but could not be taken without disturbing completely the conditions under the tent.

EFFECTIVE TEMPERATURE SCALES

It should be pointed out that the temperature, humidity and movement of air inside the tent directly affect the heat loss from only that portion of the body which is covered by the tent,[3] and that the effect of the three variables is modified by the heat insulation of the bed clothes. Therefore, neither the basic nor the normal scale of effective temperature can be assumed to apply absolutely to our clinical conditions. Qualitatively, however, a rise of effective temperature of from 1 to 6 degrees with respect to the room air, owing to an increase in humidity, or in dry bulb temperature or in both factors, was accompanied by increased discomfort and sometimes distress of the patient, even though the tent covered only the head and neck. We feel justified, therefore, in applying one of the available effective temperature scales as a guide to the thermal effect of the tent as compared to that of the outer room.

On the basic scale the effective temperatures are lower than on the normal scale because of the larger body area exposed for the basic scale determinations, resulting in greater heat loss and a greater sensation of coolness. A patient in bed is protected against heat loss as much as or more than a person normally clothed. Therefore, of the two available scales for evaluating the combined thermal effect of the three atmospheric variables, the normal scale would seem to be better adapted for clinical use. It will be used instead of the basic scale in our subsequent publications on atmospheric conditions in oxygen tents.

TENT AND METHOD OF ADJUSTMENT

The tent or hood has a collapsible frame made of two semicircular metal hoops, pivoting at the center about the oxygen inlet tube which projects about 1½ inches (3.8 cm.) below the frame, and ends in a seven-eighths inch (2.2 cm.) finely perforated spreader. The canopy is of canvas, cut in four pieces and seamed so as to form a slightly elongated umbrella with a cellophane window in front and an opening for the tube at the top. When slipped over the frame the canopy is held taut by rings hooked to the ends of the metal ribs, and encloses a volume of roughly 1½ cubic feet. The canvas projects 5 or 6 inches (12 or 15 cm.) beyond the ends of the ribs. Figure 1 is a diagram of the tent as specially adjusted for the present tests with a flow meter to measure the oxygen feed, a hygrometer and thermometer suspended from the frame, and two additional openings in the canvas for the sample of air pumped to and returned from the gas analyser.

3. The rate of heat loss from the mouth, pharynx, trachea, bronchial tree, lungs, etc., is affected by the atmospheric conditions over the mouth and nose.

For clinical use the tent is adjusted over the patient's head, and oxygen is supplied through a hospital flow regulator to the inlet tube at the top. For cooling purposes, and to make the canvas less porous, the cloth is usually moistened by occasional sponging. A flat rubber collar or folded rubber sheets are adjusted about the patient's neck under the tent to keep pillow and bedding dry. An electric fan is sometimes directed on the tent to increase the cooling effect of evaporation from the moist walls.

The factors that can be varied by the operator are the rate of oxygen flow, the frequency of sponging of the canvas and the rate of air movement around the tent. Other variables affecting the conditions in the tent include disturbances of the tent by the patient, the rate of metabolism and of elimination of moisture by the patient, and the temperature and relative humidity of the surrounding air.

EXPERIMENTAL WORK

A number of tests were carried out with and without a patient under the tent. In the tests without a patient the tent was placed on a bed protected by a rubber

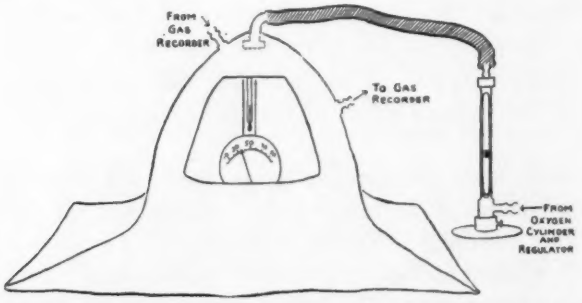


Fig. 1.—Diagram showing canvas tent, as adjusted for tests, with hair hygrometer and thermometer, tubes leading to and from oxygen and carbon dioxide recorder, oxygen inlet tube and float flow gage.

sheet. When the tent was used on a patient the bedding was protected by folded rubber sheets or rubber bibs reaching across the shoulders and behind the pillow, and the canvas flap at the base of the tent was adjusted as smoothly as possible over the rubber sheet.

The oxygen supply was controlled by means of a hospital flow regulator and a flow meter of the weighted float type (fig. 1), calibrated for oxygen in liters per minute. Oxygen and carbon dioxide concentrations were measured with the recording thermal conductivity analyser described elsewhere,[4] the gas sample being withdrawn through an opening at one side of the tent and pumped back through a second opening in the upper part of the tent at the rate of about 0.5 liter per minute. The carbon dioxide readings cannot be considered as accurate during a period of rapid change in oxygen concentration, owing to unequal oxygen concentrations in the two compartments of the carbon dioxide double by-pass cell. Since the oxygen concentration is measured in a single by-pass cell, the carbon dioxide-free sample of air being compared with an air mixture of fixed composition, the oxygen readings approximate the usual degree of accuracy of the instrument (from

4. Bullowa, J. G. M., and Lubin, Grace: J. Clin. Investigation **10**:603, 1931.

1 to 2 per cent) even during a rapid change in concentration. Allowance must, however, be made for a few minutes' lag in interpreting the readings. The oxygen values given have been corrected for the scale error of the instrument.

Temperature and relative humidity outside and inside the tent were measured with Fahrenheit thermometers reading to 2 degrees, and with hair hygrometers. The hygrometers used were obtained from Henry J. Green, Brooklyn, and were found to be unusually reliable. They were checked with a sling psychrometer before and after the tests to within 2 per cent relative humidity.

The results of seven experiments are shown graphically in figures 2 to 8, and the most important relations are roughly summarized in tables 1 to 4. Referring to the curves, the time is plotted along the horizontal axis. The method of operation is descriptively charted in the upper small sections. The rate of oxygen flow

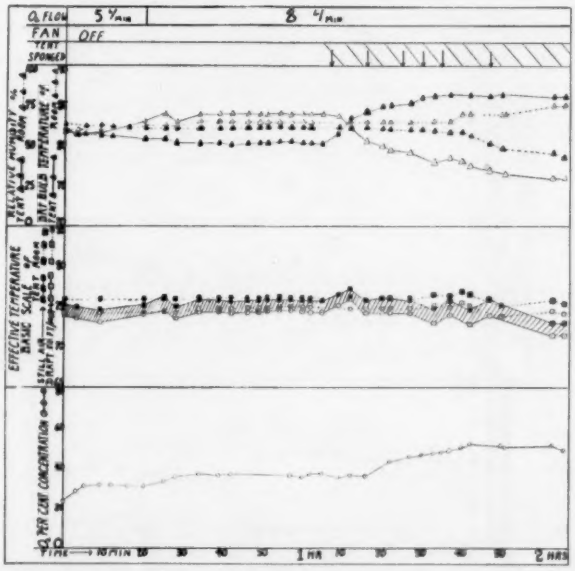


Fig. 2.—Atmospheric conditions in the canvas tent during experiment 1, with no patient. The rates of oxygen flow were 5 and 8 liters per minute. The tent was dry at the start of the test and later was moistened intermittently, as indicated by the arrows. The fan was omitted.

is given in liters per minute. The use of an electric fan for a given period is indicated by a shaded area in the small section headed "Fan." The arrows in the third small section, with the heading "Tent Sponged," refer to the time at which the tent canopy was moistened. Cross-hatching in this section refers to a moist tent, the section being left blank for the period at the beginning of some of the tests when a dry canvas canopy was employed.

The curves in the three main sections are plotted from observations of atmospheric conditions and from calculations based on these observations. In the lowest of the three main sections are given the carbon dioxide concentration (black disks) and the oxygen concentration (black circles). Dry bulb temperature and relative humidity are shown in the uppermost of the three main sections, as white and

black triangles respectively, the solid line curves representing conditions in the tent, while the broken line curves refer to conditions in the room within a few feet of the tent. In the middle section are shown the basic scale effective temperatures corresponding to these temperatures and humidity conditions for still air (black squares) and for a draft of 50 feet (15 meters) per minute (white squares). The solid line curves, connected by cross-hatching, refer to conditions in the tent, and the broken line curves to conditions in the room.

Closely parallel results are obtained when the effective temperature is calculated according to the normal scale, as can be seen from figure 9, in which the conditions in the room and in the tent for experiment 7 are represented on both the normal effective temperature scale (originally determined for men normally clothed) and on the basic scale (originally determined for men stripped to the waist).

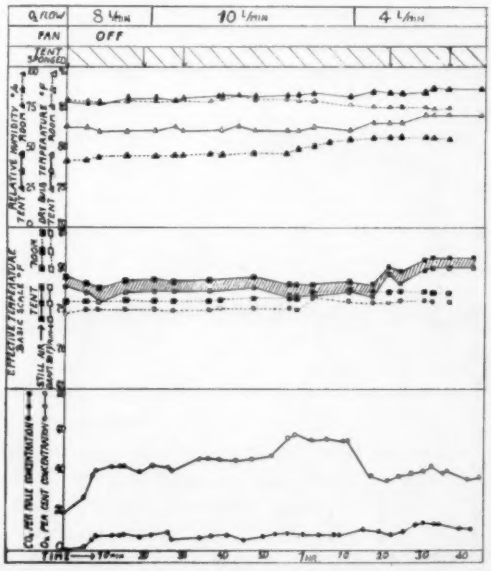


Fig. 3.—Atmospheric conditions in the canvas tent during experiment 2. The patient was a man. The rates of oxygen flow were 8, 10 and 4 liters per minute. The tent was moistened intermittently, as indicated by the arrows. The fan was omitted.

AIR MOVEMENT IN THE TENT

The effect of air movement on the basic scale effective temperature can be gaged in figures 2 to 9 by the width of the shaded band representing the probable range for the tent, as well as by the width of the band enclosed within the broken lines for probable conditions in the room. It will be seen that for the humidity and temperature conditions covered, the effective temperature on the basic scale is decreased by from 1 to 3 degrees by a draft of 50 feet per minute. On the normal scale the cooling effect of air movement is not so great, as can be seen from figure 9. A maximum draft of 50 feet per minute was chosen in computing the effective temperature, since this was about as rapid a draft

as was directed over any considerable area of the patient's body under the various conditions in the tent.

The air movement inside the tent without a patient was tested by means of a 4 inch (10.1 cm.) vane anemometer and a katathermometer [5] under various rates of oxygen flow. The intensity of the air current was found to vary with the rate of oxygen flow, and for a given flow rate to be mainly localized as a draft in the line of the oxygen current.

An appreciable vertical air current was observed directly under the oxygen inlet nozzle, even at bed level, about 1 foot (2.5 cm.) below the nozzle. As measured with the anemometer this draft amounted to about

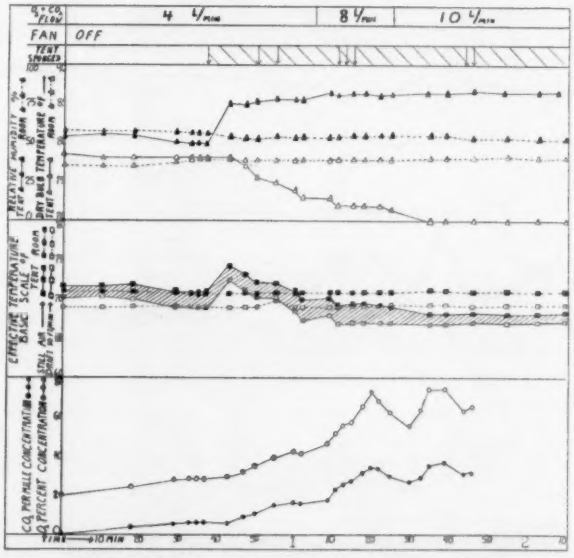


Fig. 4.—Atmospheric conditions in the canvas tent during experiment 3, with no patient. The rates of flow of the oxygen and carbon dioxide mixture (95 and 5 per cent) were 4, 8 and 10 liters per minute. The tent was dry at the start of the test and later was moistened intermittently, as indicated by the arrows.

100 feet (30 meters) per minute for a flow of 10 liters of oxygen per minute (the highest flow rate used in the present tests), to 70 feet (21 meters) per minute for a flow of 8 liters and to 30 feet (9 meters) per minute for a flow of 5 liters. The velocities for these three oxygen flow rates were slightly greater when measured only 6 inches (15.2 cm.)

5. Yaglou has questioned in private conversation with one of us (G. L.) the validity of using a katathermometer in measuring air movement in enclosed spaces. Since the air currents in the tent were below those measurable by our anemometer (from 20 to 30 linear feet in most instances) and the effective temperature scales used were for air currents between 0 and 50 linear feet, our conclusions in reference to the resultant effective temperatures found are unaffected by this criticism.

below the nozzle, being respectively 110, 90 and 50 feet (33, 27 and 15 meters) per minute. These velocities do not, however, represent the draft on the exposed portion of the patient's body, since the head and face were not vertically under the nozzle. When the anemometer was moved slightly away from the center of the tent toward one of the walls, the velocity fell rapidly. Near the walls of the tent no appreciable air motion could be detected in any direction with the anemometer, which does not indicate velocities of less than from 20 to 30 feet (6 to 9 meters) per minute.

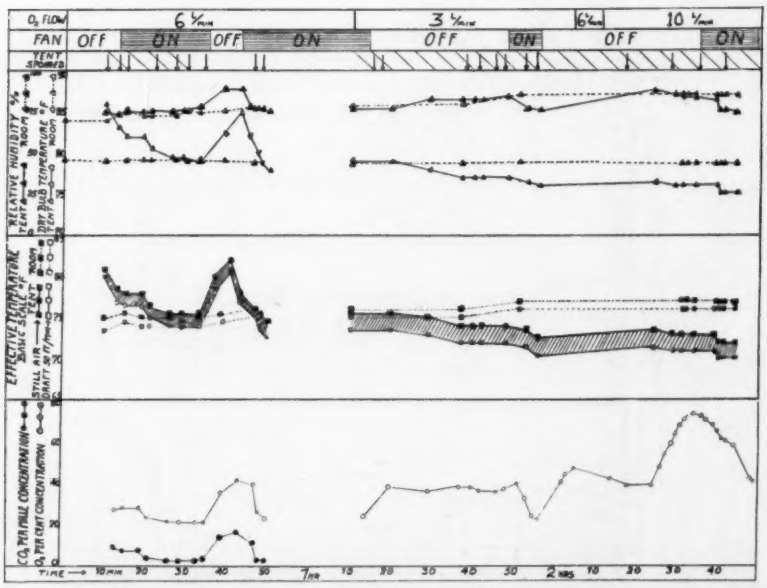


Fig. 5.—Atmospheric conditions in the canvas tent during experiment 4. A patient (a man with a rectal temperature of from 102 to 103.6 F.) was used during the first part of test, but none during the second part. The rates of oxygen flow were 6, 3, 6 and 10 liters per minute. The tent was intermittently moistened, as indicated by the arrows. The fan was turned on and off. The patient complained of unendurable heat when the fan was turned off (time: from forty to forty-three minutes); he was greatly relieved when released from the tent.

Air measurement with a katathermometer bulb placed about 8 inches (20.3 cm.) below the inlet nozzle gave readings of several hundred feet per minute when the instrument was suspended vertically under the inlet. When it was suspended 1 inch (2.5 cm.) off center, however, the air movement calculated from the cooling rate was 100 feet (30 meters) per minute or less, even at a flow rate of 10 liters of oxygen per minute, and 2 inches (5 cm.) off center the air movement at this maximum rate of flow was less than 50 feet per minute. Readings taken near the

walls of the tent were similarly less than 50 feet per minute with flow rates of 8 and 10 liters of oxygen per minute. The air movement for all rates of oxygen flow thus appears to be less than 50 feet per minute throughout the tent, except in a vertical cone below the inlet, with a cross-section of from 3 to 4 square inches (58 to 103 sq. cm.) at the base, at the very center of which a highly localized draft of several hundred feet per minute may be detected.

When the electric fan (diameter, 10 inches [25.4 cm.]) was directed against the outer walls of the tent, causing a draft of about 1,200 feet (365 meters) per minute, the draft transmitted through the walls of the

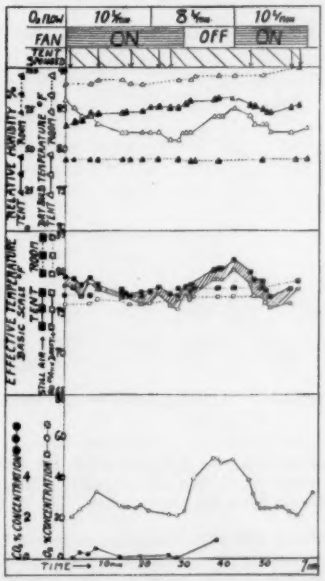


Fig. 6.—Atmospheric conditions in the canvas tent during experiment 5. The patient was a 10 year old child with a rectal temperature of from 101 to 102 F. The rates of oxygen flow were 10, 8 and 10 liters per minute. The tent was intermittently moistened as indicated by the arrows. The fan was turned on and off. The patient was uncomfortable with the fan off.

tent amounted to only about 13 feet (3.9 meters) per minute as measured by the rate of cooling of the dry katathermometer bulb, and was too small for measurement with the anemometer when the tent was dry as well as when it was wet. It thus appears safe to assume drafts varying from 0 to 50 feet per minute in the tent during the tests. A similar range of drafts can be assumed for the room. The tests were carried out during the summer and early fall in a large ward with many open windows.

ANALYSIS OF RESULTS

1. *Dry Tent.*—A dry tent was tested with and without a fan at different flow rates of oxygen in experiments 1, 6 and 7 (figs. 2, 7 and 8). In experiment 3 (fig. 4) a mixture of oxygen and carbon dioxide was substituted for the oxygen. The results of these tests are summarized in table 1.

Without a patient, the effective temperature was, of course, about the same inside as outside of the tent. With a small infant under the tent the effective temperature exceeded that in the room by from 4 to

TABLE 1.—*Atmospheric Conditions in a Dry Canvas Tent Without a Fan*

Experiment	Rate of Flow, Liters per Min.	Oxygen, per Cent	Carbon Dioxide, per Cent	△ E. T.* F.	△ D. B.† F.	Patient	Figure No.	Time From	Time To
3	Oxygen–carbon dioxide mixture (95:5%) 4	28	0.6	+1.5 to −0.2	+1.0 to +0.2	None	4	0	37 min.
1	Oxygen (100%) 5	30 to 31	...	0 to −1	−0.5 to +0.5	None	2	0	20 min.
6	Oxygen 6	33	0.2	+6 to +7	+6 to +7	D. B., 9 lb. infant	7	0	17 min.
7	Oxygen 6	29 to 33	0.1 to 0.2	+4 to +6	+5 to +7	D. B., 9 lb. infant	8	0	49 min.
1	Oxygen 8	36 to 37	...	+0.5 to −0.6	+0.5 to +1.5	None	2	20 min.	1 hr., 5 min.

* △ E. T. = difference between effective temperature in the tent and in the room, using the basic scale, and assuming a draft of 50 feet per minute. When △ E. T. is positive, the tent, as gaged by this scale, is warmer than the room; when △ E. T. is negative, the tent is cooler than the room.

† △ D. B. = difference between dry bulb temperature in the tent and in the room. When △ D. B. is positive, the thermometer reading in the tent is higher than that in the room. When △ D. B. is negative, the thermometer reading in the tent is lower than that in the room.

7 degrees,[6] but was reduced to room conditions by means of the fan. It is interesting to note from figures 6 and 7 that the excessive effective temperature in a dry tent is due to an increase in dry bulb temperature, while the relative humidity approximates that in the room.

The low concentrations of oxygen and carbon dioxide recorded under these conditions consistently attest to the inefficiency of the dry tent in retaining oxygen, and to a parallel porosity toward carbon dioxide. A concentration of 50 per cent oxygen cannot be maintained even at a flow rate of 10 liters of oxygen per minute, which represents a consumption of about two and a fourth large cylinders of oxygen in twenty-four hours. This rate is almost double that required for many tents and is equivalent to the normal oxygen needs of a well designed

6. With children and adults an even greater excess is to be expected.

oxygen chamber providing 50 per cent oxygen for two patients and a nurse.

When a dry tent is operated near an electric fan (fig. 8, from forty-nine minutes to one hour and three minutes) the oxygen escapes so rapidly that no excess concentration can be maintained with a flow of 6 liters per minute. In this condition the tent is quite useless.

2. *Moistened Tent: Tests Without a Patient.*—In experiments 1 and 3 and during the latter part of experiment 4 a moistened tent was tested without a patient. The results may be found in figures 2, 4 and 5, and are roughly summarized in table 2. It will be seen that

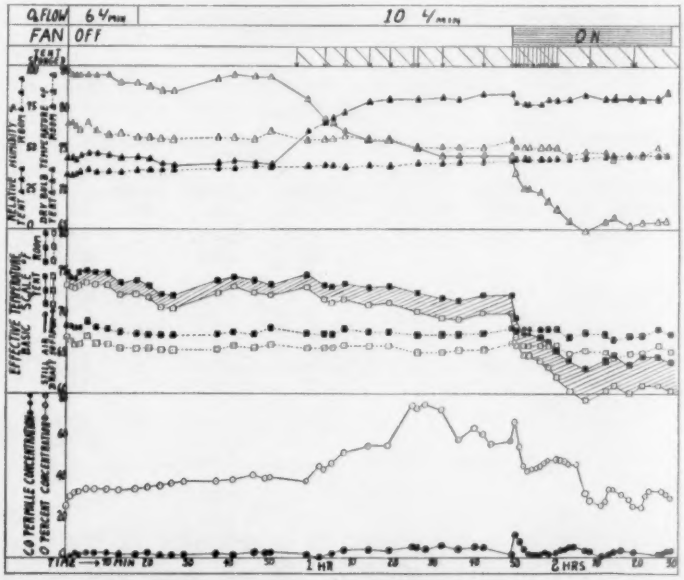


Fig. 7.—Atmospheric conditions in the canvas tent during experiment 6. The patient was a 9 pound (4 Kg.) infant, aged 4 months. The rates of oxygen flow were 6 and 10 liters per minute. The tent was dry at the start of the test and later was moistened intermittently and for ten minutes continously, as indicated by the arrows. The fan was turned on and off.

the dry bulb temperature is markedly depressed (from 5 to 11 degrees) by evaporation of moisture from the walls, whereas the effective temperature of the tent air is only moderately depressed (from 0 to 5 degrees) because of the pronounced increase in relative humidity. This is a clear example of the lack of correspondence between dry bulb temperature and effective temperature. If the effective temperature is a true index of the thermal effect of the environment on the body, the dry bulb thermometer alone is obviously not sufficient as a guide and may be distinctly misleading if used without a hygrometer, as will be more

conclusively shown by tests on the tent under normal working conditions, with a patient.

When used without a fan, the moistened tent is much less porous than the dry tent, a concentration of at least 50 per cent oxygen being maintained in the empty tent with a flow of 8 liters per minute (table 2). Even under these conditions, however, it is more wasteful of oxygen than properly designed tents many times larger and roomier, made of rubberized fabric and provided with artificial ventilation.

The effect of a fan on a moistened tent without a patient can be seen from figure 5 (one hour and forty-eight minutes to one hour and fifty-six minutes and two hours and thirty-five minutes to two hours and forty-nine minutes). The effective temperature in the tent is slightly

TABLE 2.—*Atmospheric Conditions in a Moist Canvas Tent Without a Fan and With No Patient*

Experiment	Rate of Flow, Liters per Min.	Oxygen, per Cent	Carbon Dioxide, per Cent	△ E. T.* F.	△ D. B.* F.	Figure No.	Time	
							From	To
4	Oxygen 3	36 to 38	...	—1.5 to —4.0	—6.8 to —9.0	5	1 hr., 20 min.	1 hr., 48 min.
3	Oxygen-carbon dioxide mixture (95:5%) 4	41 to 42	1.6	0 to —1.8	—4.8	4	37 min.	1 hr., 5 min.
1	Oxygen 8	50 to 52	...	—0.6 to —2.3	—7 to —9	2	1 hr., 7 min.	2 hr.
4	Oxygen 10	73	...	—5	—11	5	2 hr., 11 min.	2 hr., 35 min.

* See table 1.

lower than without a fan, owing to a reduction in both dry bulb temperature and relative humidity. At the same time the oxygen concentration is greatly reduced, from 39 to 22 per cent at 3 liters per minute, and from 73 to 41 per cent or less at 10 liters per minute.

3. *Moistened Tent: Tests With a Patient and Without a Fan.*—The results obtained with patients in a moistened tent are roughly summarized for conditions without a fan in table 3 and with a fan in table 4. Without a fan an oxygen flow of 6 liters per minute appears ample to maintain a concentration of slightly more than 40 per cent. Tests with 8 liters per minute for short periods gave concentrations of between 38 and 48 per cent, and a flow of 10 liters per minute gave concentrations of more than 50 per cent. These results agree with those in table 2 for an empty moist tent without a fan, and indicate that at a high rate of flow the partial pressure of oxygen can be maintained at a therapeutically active level for pronounced degrees of anoxemia.

A study of the temperature conditions shows a pronounced change from the conditions in the empty tent due to the heat and moisture given out by the patient, and offers further evidence for the fallacy of judging the conditions by dry bulb temperature alone. It will be seen from table 3 that although the dry bulb temperature in the tent is not so strongly depressed as in the tests without a patient, it nevertheless remains at or below room conditions, while the effective temperature, on the other hand, is from 1 to 6 degrees higher inside the tent than in the room. As gaged by dry bulb temperature alone the air in the tent would appear to be cooler than that in the room. As gaged by effective

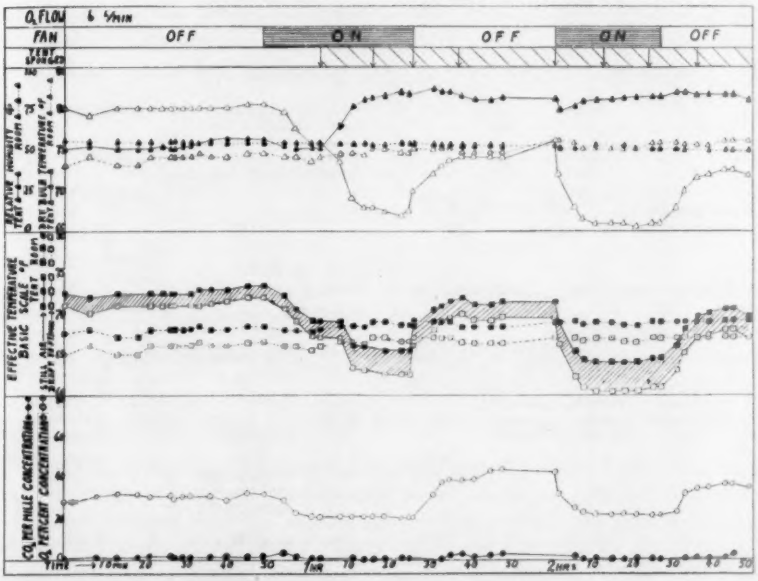


Fig. 8.—Atmospheric conditions in the canvas tent during experiment 7. The patient was a 9 pound infant, aged 4 months. The rectal temperature at the start of the test was 98.6 F. The rate of oxygen flow was 6 liters per minute. The tent was dry at the start of the test and later was moistened intermittently, as indicated by the arrows. The fan was turned on and off.

temperature, the air in the tent should feel actually warmer than the room air. As a matter of fact, whenever the fan was turned off, the patients (adults in tests represented in figures 3 and 5 and a child in the test shown in figure 6) became restless and complained of the heat.

4. *Moistened Tent: Tests With a Patient; Fan in Use.*—The results of tests with a patient in a moistened tent under a fan are shown in figures 5, 6, 7 and 8, and are summarized in table 4. It will be seen that the increased evaporation rate lowers the dry bulb temperature in the tent to from 3 to 10 degrees below room temperature. The effective temperatures observed were also lower than those without a fan,

TABLE 3.—*Atmospheric Conditions in a Moist Canvas Tent, With Patient and Without Fan*

Experiment	Rate of Flow, Liters per Min.	Oxygen, per Cent	Carbon Dioxide, per Cent	△ E. T.* F.	△ D. B.* F.	Patient	Figure No.	Time From	Time To
2	4	36 to 41	0.8 to 1.3	+2.2 to +4.2	−2 to −0.8	T. H., adult male	3	1 hr., 12 min.	1 hr., 44 min.
4	6	41	1.6	+6	0	J. S., adult male; rectal temperature, 102-103.6 F.	5	35 min.	43 min.
7	6	42 to 44	0.3	+3.7 to +3.0	−0.6 to 0	D. B., 9 lb. infant; rectal temperature, 98-99 F.	8	2 hr., 27 min.	2 hr., 49 min.
2	8	38 to 42	0.7	+1.3 to +2.3	−3.3 to −3.9	T. H., adult male	3	0	21 min.
5	8	46 to 48	...	+3†	−4†	L. W., 10 yr. old child; rectal temperature, 101-102 F.	6	29 min.	42 min.
2	10	40 to 57	0.5 to 0.9	+2.4 to 0	−4 to −3.2	T. H., adult male	3	21 min.	1 hr., 12 min.
6	10	43 to 75	0.3 to 0.6	+5.7 to +3.7	0 to −1	D. B., 9 lb. infant	7	56 min.	1 hr., 49 min.

* See table 1.
† Steady state not reached.

TABLE 4.—*Atmospheric Conditions in a Moist Canvas Tent With a Patient and With a Fan*

Experiment	Rate of Flow, Liters per Min.	Oxygen, per Cent	Carbon Dioxide, per Cent	△ E. T.* F.	△ D. B.* F.	Patient	Figure No.	Time From	Time To
4	6	20.5	0.3	+2 to −0.5	−3 to −5.7	J. S., adult male; rectal temperature, 102 F.	5	13 min.	35 min.
4	6	23.0	0.3	0 to −2†	−5 to −6	J. S., adult male; rectal temperature, 102-103.6 F.	5	43 min.	50 min.
7	6	20.5	0 to 0.1	−3.5 to −4.5	−7 to −7.5	D. B., 9 lb. infant; rectal temperature, 98-99 F.	8	1 hr., 3 min.	1 hr., 26 min.
7	6	20.5 to 21.0	0	−6 to −6.5	−9 to −9.7	D. B., 9 lb. infant; rectal temperature, 98-99 F.	8	2 hr., 1 min.	2 hr., 27 min.
5	10	25.0 to 32.0	0 to 0.5	+2.5 to 0	−6.5 to −6	L. W., 10 yr. old child; rectal temperature, 101 F.	6	0	21 min.
5	10	20.5 to 26.0	...	0 to −1.8	−6 to −8	L. W., 10 yr. old child; rectal temperature, 102 F.	6	42 min.	1 hr.
6	10	25.0 to 33.0	0 to 0.3	−3.0 to −6.0	−7 to −9.5	D. B., 9 lb. infant	7	2 hr.	2 hr., 28 min.
6†	10	42.0 to 48.0	0.2	‡	‡	D. B., 9 lb. infant	7	1 hr., 49 min.	2 hr.

* See table 1.
† Steady state not reached.
‡ Tent continuously sponged.

varying from 2.5 degrees above room temperature to 2 degrees below for the adults and the child tested, and from 3 to 6.5 degrees below room temperature for the infant.

The findings with respect to oxygen concentration agree with those in similar tests with an empty tent, and point to a rapid escape of oxygen through the canvas under the action of the fan. With a flow rate of 6 liters of oxygen per minute and the usual intermittent sponging, the concentration in the tent was practically that of ordinary air, while with a rate of 10 liters per minute it remained at from 21 to 33 per cent. In experiment 6 (fig. 7) when the drying action of the fan was counteracted by *continuous* sponging of the canopy, a concentration of between 40 and 50 per cent was attained at a feed of 10 liters per minute. As can be seen from figure 7, the oxygen concentration fell rapidly when the sponging was interrupted, rising slightly after each subsequent sponging.[7]

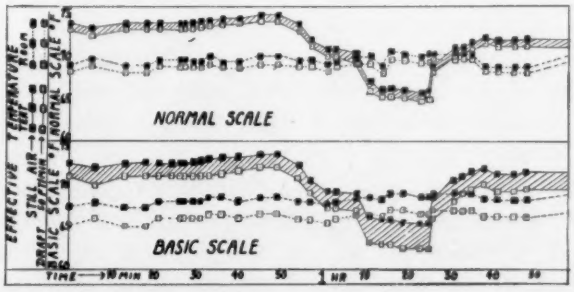


Fig. 9.—A comparison of normal and basic scale effective temperature curves, calculated from temperature and humidity conditions in the tent (solid lines) and in the room (broken lines) in experiment 7. The effect of air movement is indicated by the width of the band between the curves for still air (black squares) and the curves for a draft of 50 feet per minute (white squares), the cooling effect of the draft being greater according to the basic scale than according to the normal scale. The effective temperatures are higher when interpreted according to the normal scale, but the relative difference between tent and room conditions is about the same for both scales.

The double effect of the fan in depressing the effective temperature to tolerable values on the one hand, while lowering the oxygen content to the level of ordinary room air on the other hand, can be seen especially clearly in figure 8. In this condition the tent is obviously quite valueless for the purpose of increasing the partial pressure of oxygen in the inspired air.

7. The apparent lag of a few minutes in the response of the oxygen concentration to the time of moistening is characteristic of the thermal conductivity recorder used for analysis.

CONCLUSIONS

1. Canvas is not a suitable material to confine oxygen because when the tent is dry it is too porous; it fails to confine oxygen but confines body heat. Sealing the pores with water increases the retention of oxygen, but also causes an increase in the humidity and heat retention.

2. A tent which depends for the circulation of air on the currents produced by oxygen entering from an aperture on the top is unsatisfactory because of the insufficient circulation of air.

3. The use of a fan for cooling by evaporation from a canvas tent is an inadvisable procedure because the rate of depletion of oxygen is increased unless the tent is continuously wet.

THE MORO REFLEX IN THE NEW-BORN

HEYWORTH N. SANFORD, M.D.
CHICAGO

Moro [1] first described the embrace reflex that bears his name. He found that when an infant is placed on a table and then the table is forcibly struck on either side of the child, a motor reaction is obtained. The arms are suddenly thrown out in an embrace attitude, describing an arc and tending to approach one another with a slight tremor. The fingers are at first spread and then closed. This observation has been confirmed by Freudenberg,[2] Schaltenbrand,[3] Peiper and Isbert [4] and Gordon.[5]

Gordon's observations on fifty-four new-born infants are of interest. He found that forty-seven gave a positive reaction. The intensity of the reaction seemed to be influenced by the general condition of the child. In two cases of cerebral hemorrhage a feeble response was obtained on the first two days, with improvement as the general condition became better. Of two other infants with intracranial hemorrhage one gave a weak and the other a negative response, but both improved later. He also observed four infants with unilateral brachial palsy. The reflex was released on the normal, but not on the affected, side. Improvement in the palsy was accompanied by a gradually increasing response on the affected side, as evidenced in one child who was observed for several months.

During the past three years the Moro reflex has been tested on 1,634 new-born infants for the first ten to fourteen days of life. There were forty-eight cases in which it was absent symmetrically for various periods, and ten in which it was asymmetrical.

Certain conditions are necessary for satisfactory testing of the Moro reflex in the new-born. The child should be quiet; while the reflex is

---

From the Department of Pediatrics, Rush Medical College of the University of Chicago, and the Presbyterian Hospital.

1. Moro, E.: Das erste Trimenon, München. med. Wchnschr. **65**:1147, 1918.
2. Freudenberg, E.: Der Morosche Umklammerungsreflex und das Brudzinskische Nachenzeichen als Reflexe des Säuglingsalters, München. med. Wchnschr. **68**:1646, 1921.
3. Schaltenbrand, G.: Normale Bewegungs- und Lagereaktionen bei Kindern, Deutsche Ztschr. f. Nervenh. **87**:23, 1925.
4. Peiper, A., and Isbert, H.: Ueber die Körperstellung des Säuglings, Jahrb. f. Kinderh. **115**:142 (Feb.) 1927.
5. Gordon, M. B.: The Moro Embrace Reflex in Infancy: Its Incidence and Significance, Am. J. Dis. Child. **38**:26 (July) 1929.

usually obtained in a crying child, it sometimes is not. The method of obtaining the reflex is of importance. When the method described by Moro is used, the table must be so constructed that a change of position is produced by the blow. For this reason a solid-topped wooden table or one made of porcelain or iron is useless. This was not realized until another nursery was added to the hospital, which was equipped with a wooden examining table apparently similar to that previously used. It was impossible to obtain a satisfactory reflex until a supporting beam under the top had been removed, thus making it similar to the original table. In the absence of such a table, an even more satisfactory result may be obtained by holding the feet and head firmly in a horizontal position and allowing the head and upper part of the

A, a normal new-born infant, showing symmetrical Moro reflex. *B*, fracture of the right clavicle in a new-born infant, showing asymmetrical Moro reflex. *C*, birth paralysis of the left arm in a new-born infant, showing asymmetrical Moro reflex; note that the forearm is not flexed on the upper arm as in the case of fracture of the clavicle.

body to drop a few inches. Obviously this method is not the one of choice if a suitable table is available. Figure *A* illustrates a normal new-born infant showing a typical reaction to the Moro reflex.

In thirty-nine of forty-eight cases of cerebral affections in the new-born, the condition was called cerebral edema for want of a better name. All of these cases have several common characteristics: First, the Moro reflex was absent on the first day and gradually returned during the following four days. Of the thirty-nine cases, it was present to a greater or less extent in fourteen on the second day, in twenty-seven on the third day, and in the remaining two on the fifth day. All of these infants had been born after a long, difficult labor, or by a forceps delivery. Physically they all had some caput succedaneum,

wide sutures or full fontanels. During the first three days they manifested signs of intracranial pressure, such as attacks of cyanosis, failure to nurse, vomiting and twitchings. Several were thought to have a cerebral hemorrhage, but the spinal fluid was normal on cisternal puncture. All were perfectly normal by the fifth day and left the hospital in good condition.

There were seven cases of cerebral hemorrhage. In six of these cases the reflex was present on the first day; in the remaining case it was absent on the first day. It returned in two cases on the sixth day, in one case on the seventh day, in two cases on the eighth day and in one case on the tenth day. The last patient died on the eleventh day, and autopsy showed a tentorial hemorrhage. The six remaining

TABLE 1.—*Conditions Causing a Symmetrical Absence of the Moro Reflex in the New-Born*

Diagnosis	Day										Total Cases
	1	2	3	4	5	6	7	8	9	10	
Cerebral edema	39	25	12	2	0	0	0	0	0	0	39
Cerebral hemorrhage	1	6	7	7	7	5	4	3	3	2	7
Anencephaly	1	1	1	Died	..	..	..	..	..	..	1
Hydrocephalus and spina bifida	1	1	1	1	1	Died	..	..	..	..	1

TABLE 2.—*Conditions Causing an Asymmetrical Absence of the Moro Reflex in the New-Born*

Diagnosis	Day										Total Cases
	1	2	3	4	5	6	7	8	9	10	
Fracture of clavicle	8	8	8	8	8	6	5	3	2	2	8
Birth paralysis unilateral	2	2	2	2	1	1	1	1	1	1	2

patients left the hospital in good condition. The diagnosis of cerebral hemorrhage was made on the basis of the physical findings of tense fontanel, cyanosis, convulsions and flaccidity or spasticity plus a spinal fluid obtained by cisternal puncture which contained blood and was under pressure. With one exception, all of these patients showed a positive Moro reflex within the first twelve hours of life, which then disappeared and returned as the infant's condition improved. In none of them was there a return before the sixth day.

Six of the ten cases of asymmetrical Moro reflex cited in table 2 have been previously reported.[6] In this condition the infant gives a positive reaction on the unaffected side, but on the side of the injury there is no response, although the forearm is flexed on the upper arm (fig. *B*). As the callus is formed, the reflex returns, being weakly positive in from six to ten days.

6. Sanford, H. N.: The Moro Reflex as a Diagnostic Aid in Fracture of the Clavicle in the New-Born Infant, Am. J. Dis. Child. **41**:1304 (June) 1931.

In the two cases of unilateral brachial birth paralysis, a positive reaction was obtained on the uninjured side, while the affected side gave no response. It will be observed in the photograph (fig. *C*) that there is no flexion of the forearm as in fracture of the clavicle. As the condition improves, the response appears on the injured side. In one case it was weakly positive in six days.

The many conjectures as to the cause and reflex center of this phenomenon have been amply discussed by Gordon and will not be repeated here. Histologic sections of the brain of the anencephalic infant and of the child with cerebral hemorrhage give some clue as to the inhibitory tracts and will be reported on later.

The conclusions, therefore, may be summarized as follows: The Moro reflex was studied in 1,634 new-born infants during the first ten days of life. It was positively symmetrical in 1,576 cases. Forty-eight cases gave negative responses symmetrically. Of these, thirty-nine were classified as cases of cerebral edema. Cerebral hemorrhage was not proved in any of these cases, and the reflex was absent at birth, returning by the fifth day. Seven cases in which the reflex was symmetrically absent were classified as instances of cerebral hemorrhage. In one of these cases the diagnosis was proved by autopsy, and the remaining six were diagnosed by examination of the spinal fluid and physical findings. In six of these the reflex was present on the first day, but absent on the second day, until the child's condition began to improve. Generally, therefore, a symmetrical absence of the Moro reflex indicates a cerebral disturbance in the new-born. From the findings reported it appears as if in simple edema the reflex is absent at the outset and appears sooner than in cerebral hemorrhage. In cerebral hemorrhage it is present for a time (usually twelve hours), then disappears and returns more slowly. The Moro reflex, therefore, might serve as a diagnostic aid in distinguishing true cerebral hemorrhage from simple edema.

In ten cases, the reflex was absent asymmetrically. Eight of these cases were due to a fractured clavicle, and two to birth paralysis. The Moro reflex normally being symmetrical, the absence of the reflex on one side indicates either a motor paralysis or an injury to that side.

952 North Michigan Avenue.

Case Reports

GONOCOCCAL ENDOCARDITIS

IN AN INFANT TEN DAYS OLD WITH GONORRHEAL CONJUNCTIVITIS

W. W. BRANDES, M.D.
DALLAS, TEXAS

Attention has been called to the infrequent occurrence of acute ulcero-vegetative endocarditis in infants by Osler, Still, Finkelstein, Holt and others. Bass [1] cited a case reported by Czerny in an infant 1 month of age and Bidone's case of an infant that lived nineteen hours. In 1930, Sansky and Larson [2] reviewed the literature, and found the reports of five cases in infants under 7 months of age. They were unable to confirm the report of Czerny's case in the literature. In the case they reported, that of an infant 5 weeks old, the mitral valve was involved. They considered their case the first authentic one occurring in an infant during the first six weeks of life. De Vecchi [3] studied the heart valves in children under 10 years of age. In performing 1,062 autopsies he found 18 cases, 2 of which were in infants under 1 year. He stressed the point that gross lesions may not be present, and yet microscopic lesions may be found on careful search. Microscopic lesions were present in the valves in children having infections elsewhere. De Vecchi expressed the belief that the valvular lesions are characteristic, and that thrombotic masses are formed secondarily. He thinks that the latter are not so likely to form in a child under 3 years of age because of the constitution of the blood. Grant, Wood and Jones [4] consider that the small thrombi which form over small areas of degeneration and superficial necrosis on the valve are primary and offer a foothold for bacteria present in the blood stream.

Small areas of necrosis or degeneration may be due to toxic substances and not to bacteria. One must keep in mind a distinction between toxic and infectious endocarditis.

From the Pathology Laboratory, Baylor University College of Medicine.

1. Bass, M. H., in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 4, p. 362.

2. Sansky, J. M., and Larson, L. M.: Acute Bacterial Endocarditis in Infancy, Am. J. Dis. Child. **39**:1261 (June) 1930.

3. de Vecchi, Bindo: Endocarditic Processes in Childhood, Arch. Path. **12**:49 (July) 1931.

4. Grant, R. T.; Wood, J. E., and Jones, T. D.: Heart Valve Irregularities in Relation to Subacute Bacterial Endocarditis, Heart **14**:247, 1927.

REPORT OF CASE

History.—An infant, 10 days of age, weighing 5 pounds (2,267 Gm.), whose birth was slightly premature (about the eighth month), entered the Bradford Memorial Hospital, the service of Dr. W. H. Bradford. Birth was by face presentation, and the bag of waters ruptured about twelve hours previous to delivery. The mother stated that the infant was blue. On the third day an exudate was noticed in both conjunctivae. Smears from the exudate revealed intracellular and extracellular diplococci that reacted negatively to Gram's stain and had the mor-

Fig. 1.—Vegetation on the tricuspid value. Patent ductus arteriosus.

phologic characteristics of gonococci. The conjunctivitis abated under treatment; however, the infant lost weight and had a temperature of from 99 to 103 F. There was a leukocytosis of 16,500 cells. The heart beat was rapid, but no murmurs were heard. The mother was 33 years of age. This was her first pregnancy. A profuse vaginal discharge developed following delivery; smears of this material revealed gram-negative intracellular and extracellular diplococci. There was no evidence of metastatic lesions in the mother. The Wassermann test of the mother's blood gave a reaction of 4 plus.

Necropsy revealed an undernourished white girl without obvious signs of prematurity. The skin was slightly icteric. The conjunctivae were diffusely reddened and lusterless over the palpebral portion; that portion in the sulcus was

somewhat thickened. The thymus was small. The heart weighed 15 Gm. The foramen ovale was patent for 3 mm. The ductus arteriosus was widely patent throughout. There was a large yellowish-gray, friable, thrombotic mass (6 by 12 mm.) attached to the posterior leaflet of the tricuspid valve. This leaflet was partly ulcerated. Several small (1 mm.) reddened, dull areas were present on the other leaflets of the tricuspid valve. The other valves of the heart were free from gross change. The wall of the right ventricle measured from 2 to 3 mm. in

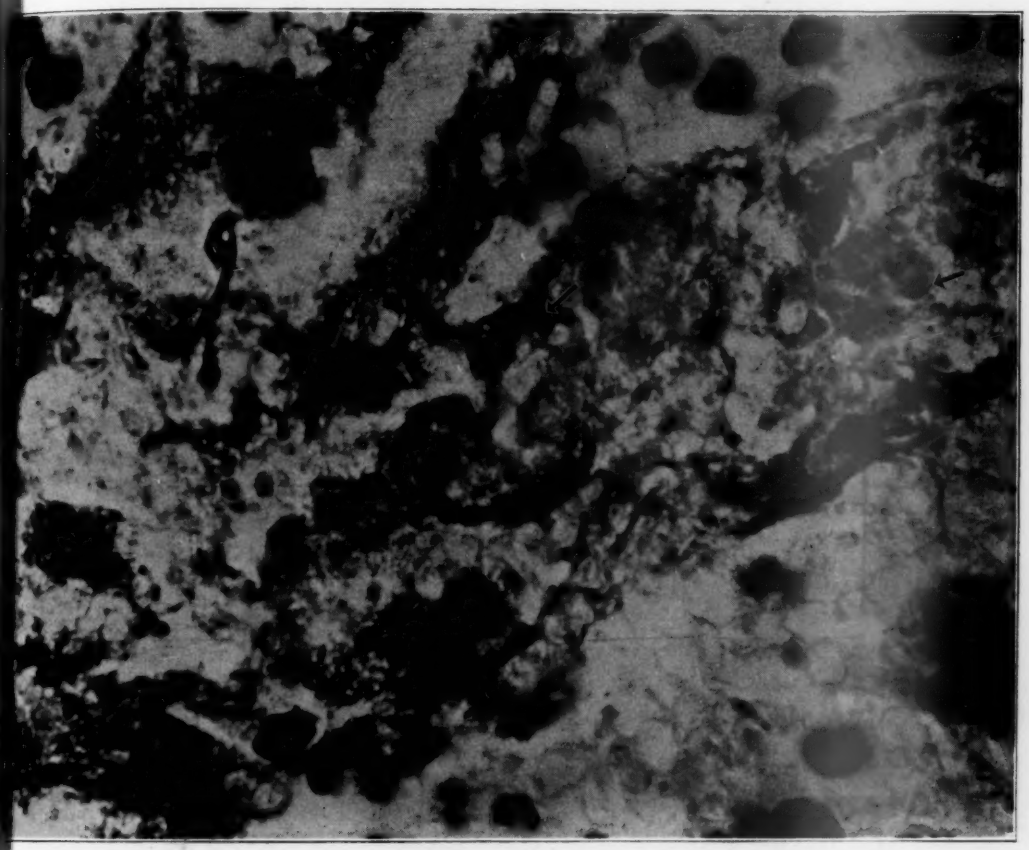


Fig. 2.—Gram-negative intracellular and extracellular diplococci in vegetation; reduced from a magnification of × 1200.

thickness, that of the left from 2 to 4 mm. There were several reddish-gray, roughly triangular subpleural areas in both lungs. The spleen was slightly large and moderately firm; it retained its shape. Permission for examination of the head was not granted.

Histologic examination of the vegetations on the tricuspid valve showed a moderate amount of fibrin in irregular strands and a large amount of débris, with a moderate number of polymorphonuclear cells. Careful search revealed gram-negative diplococci in widely scattered areas. There was a slight infiltration of

the myocardium with polymorphonuclear and mononuclear cells in scattered areas of the perivascular tissues. The reticulum of the spleen was prominent. There were small areas of hemorrhage and a slight infiltration of polymorphonuclear cells, as well as areas of partial atelectasis, in the lungs.

COMMENT

The evidence that this case is one of gonococcal endocarditis seems fairly sufficient, although a cultural test for the organisms was not made. In the first place, infection of the eyes by the gonococcus during delivery becomes evident as an active conjunctivitis between the second and the fourth day, whereas cases due to other types of organisms become manifest several days later, as a rule. The positive observations in the exudate and the presence of gram-negative diplococci in the vegetation without any other types of organisms make the etiology fairly definite.

The large size of the vegetation indicates that it may have taken more than six or seven days to form. Examination revealed it to be of recent formation. There is the possibility of intra-uterine infection through the placenta; however, there is no evidence of this. There were no signs of infection in the mother which would be expected in the form of arthritis or endocarditis if the organisms were present in the blood stream. Furthermore, endocardial involvement by one route and conjunctival involvement by another would be unusual. If the lesion on the heart valve had been of much longer duration, there might have been more infiltration and less uniformity. It would have been less friable throughout, and possibly some organization would have been evident. Numerous sections of the vegetation have been made.

I have been unable to find the report of a case of gonococcal endocarditis secondary to conjunctivitis. Available textbooks on ophthalmology fail to mention it as a complication. It is true that cases in which a necropsy was not performed may have been overlooked. Secondary involvement of the eye is stressed, and that of the meninges has been mentioned. Lucas [5] (1899) reported a series of 23 cases of arthritis following ophthalmia neonatorum. Arthritis secondary to conjunctivitis has also been mentioned by Fuchs.[6]

The localization of this lesion may be associated with the patent ductus arteriosus. In an analysis of cases of cardiac anomalies, Abbot [7] found that endocarditis develops in 22 per cent of the cases of patent ductus arteriosus. In such cases, the vegetations are frequently located

5. Lucas, Clement: Lancet **1**:230, 1899.

6. Fuchs, Ernst: Textbook of Ophthalmology, ed. 6, Philadelphia, J. B. Lippincott Company, 1919, p. 156.

7. Abbot, Maude: On the Incidence of Bacterial Inflammatory Processes in Cardiovascular Defects and on Malformed Semilunar Cusps, Ann. Clin. Med. **4**:189, 1925.

on the wall of the pulmonary artery. The increased thickness of the wall of the right ventricle may be associated with the patent ductus in the case herein reported.

Gonococcal endocarditis is found most frequently on the left side, but Thayer [8] has shown that there is a slightly greater tendency toward localization on the right than when other types of organisms are present. Kirkland [9] found that the lesions were on the right side alone in 9.65 per cent of all cases; on both sides in 9.65 per cent, and on the left side, in 80.7 per cent. The child in the case reported is the youngest patient in whom gonococcus was the etiologic agent; heretofore the cases reported have been secondary to genital infections.

CONCLUSIONS

1. A case of gonococcal endocarditis of the tricuspid valve in an infant 10 days old is reported.

2. In all probability, the endocarditis was secondary to gonorrheal conjunctivitis.

3. There was an associated patent ductus arteriosus, which may have been associated with the development and localization of the lesion.

4. The child in this case is the youngest patient having bacterial endocarditis and also the youngest patient having gonococcal endocarditis reported.

5. A report of endocarditis secondary to conjunctivitis could not be found in the literature.

---

8. Thayer, W. S.: On Cardiac Complications of Gonorrhea, Bull. Johns Hopkins Hosp. **33**:361, 1922.

9. Kirkland, H. B.: Gonococcus Endocarditis, Am. Heart J. **7**:360 (Feb.) 1932.

RUPTURE OF THE GASTROCOLIC OMENTUM AS A COMPLICATION OF WHOOPING COUGH

JOHN MILAN FRAWLEY, M.D.
FRESNO, CALIF.

The complications of whooping cough are numerous and varied. Rarely, however, does the rupture of an abdominal viscus occur during this disease as the result of severe coughing. The following case is an illustration of this unique complication.

REPORT OF CASE

History.—In consultation with Dr. Charles A. James, I saw a poorly nourished girl, 2 years of age, in the third week of an attack of whooping cough. Two days previously she had an unusually severe attack of coughing which terminated in a convulsion. Following the convulsion the child remained pale, weak and restless. The temperature of the outside atmosphere at this time, midsummer, reached a daily maximum of about 110 F. (43.3 C.). This fact was significant, because fluids given by mouth were not retained, and the child became dehydrated and presented the symptoms of high intestinal or pyloric obstruction. The temperature rose to 105 F. (40.5 C.). The white blood cells numbered 22,000, of which 50 per cent were polymorphonuclears, 46 per cent, small lymphocytes, 1 per cent, large lymphocytes and 3 per cent, transitionals.

Examination.—There was a tympanitic swelling the size of an orange in the left upper quadrant of the abdomen. No peristaltic waves were visible. Fluids given by mouth were not immediately regurgitated but increased the size of the swelling. When the stomach tube was passed and the gastric contents withdrawn, the tumor disappeared.

The patient was removed to the hospital, where parenteral fluids were administered and the general condition improved considerably. Roentgenologic examination, however, showed that barium sulphate was retained indefinitely in the stomach, and none passed through the pylorus. Accordingly, four days after the onset of symptoms an exploratory operation was advised, and, with the patient under ether anesthesia, the abdominal cavity was opened by Dr. Clinton D. Collins.

Operation and Course.—The stomach was found to be dilated and flaccid. There was a constricting band extending in the longitudinal axis of the body from the lesser to the greater curvature of the stomach. The constricting band was formed by the border of an opening in the gastrocolic omentum along the proximal half of the curvature. The opening or rupture was 3 inches (7 cm.) in length and extended well up through the gastrosplenic omentum to the cardiac end of the stomach. It followed closely the greater curvature and was practically a splitting of the peritoneal coat of the stomach at the attachment of the greater omentum.

Hernia of the cardiac half of the stomach had taken place through this opening. The herniation of the proximal half had divided the stomach into two pouches with nothing passing into the distal pouch. The proximal part was distended with gas

and liquid. It was the traction caused by the hernia which gave the stomach the appearance of being traversed by a constricting band.

The hernia was reduced, and the opening was sewed up with interrupted catgut sutures. There was some thickening of the pylorus muscle, and an incision was made in the peritoneal and muscular coats down to the mucosa according to the technic of the Fredet-Rammstedt operation. Following the operation the stomach still failed to empty. Fluids taken by mouth were not regurgitated but remained,

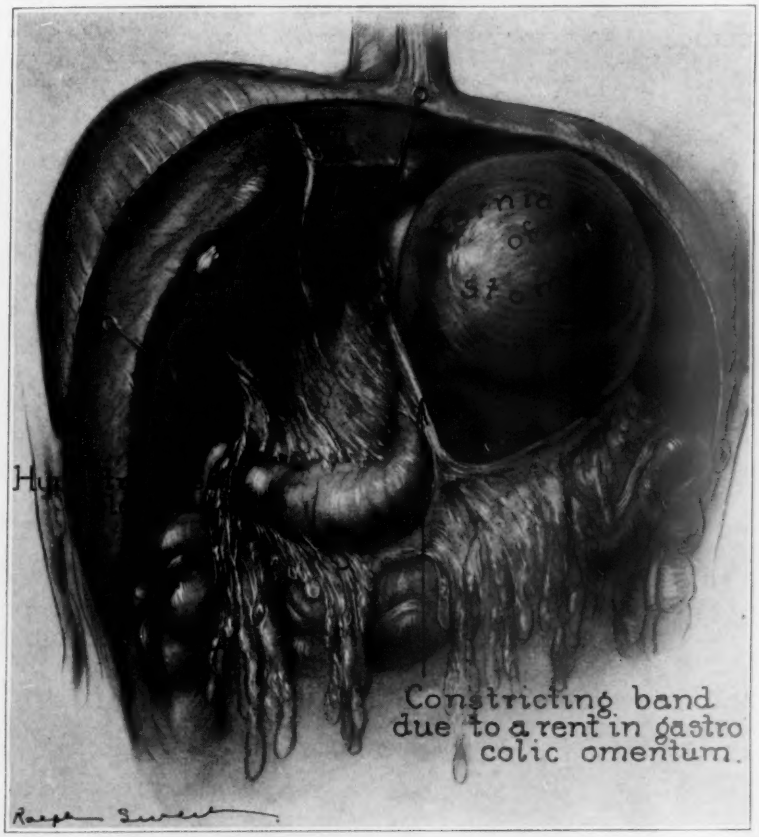


Fig. 1.—Diagram of gastric hernia through a rupture in the great omentum which occurred during an attack of whooping cough.

as before the operation, in the stomach, distending it until withdrawn by stomach tube.

Coughing spells occurred at intervals. They were severe and occasionally terminated in convulsions. From one of these convulsions the child failed to rally, and death occurred approximately twenty-four hours after operation.

Autopsy.—Postmortem examination was made through the abdominal wound. There was no free fluid in the abdominal cavity. The stomach was thick-walled, edematous and flabby. The omentum along the greater curvature of the stomach was hemorrhagic, and the edges of the rupture along the gastric border were in apposition.

When the stomach was opened, the mucous lining was seen to be markedly injected. The gastric contents consisted of a considerable quantity of a thick putty-like barium mixture. No barium sulphate had passed through the pylorus into the duodenum.

Summary.—The history and the local findings indicate that during an attack of whooping cough rupture of the gastrocolic omentum had occurred along the greater curvature of the stomach. Through this opening the stomach herniated, with resulting obstruction. At operation the hernia was reduced, but the stomach did not

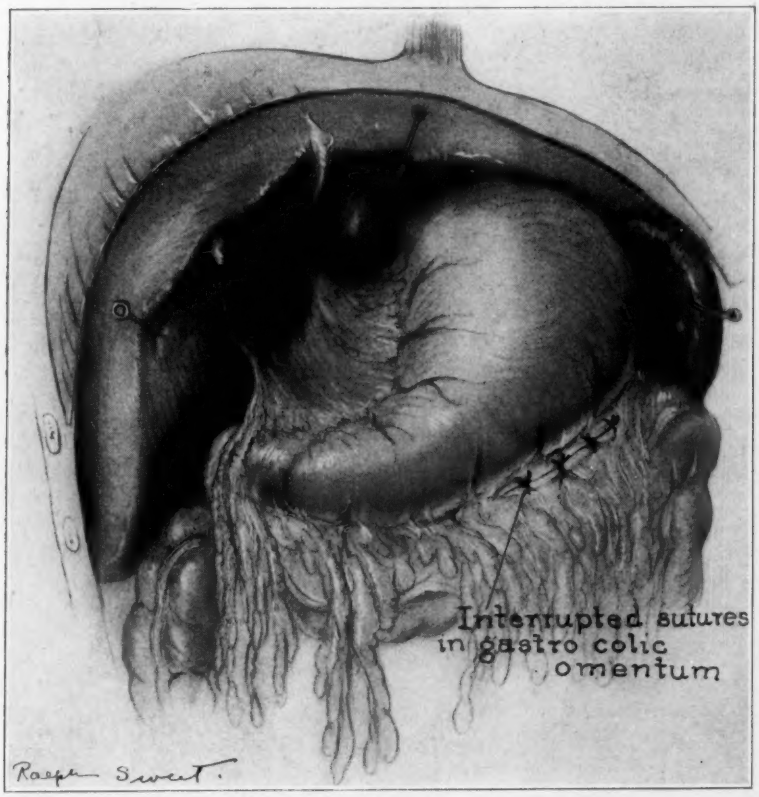


Fig. 2.—Diagram showing the flabby atonic stomach after the reduction of the hernia.

regain its tone and remained in a condition of ileus. Death occurred twenty-four hours after operation.

COMMENT

Two conditions are represented in the lesion described: first, rupture of the gastrocolic mesentery with herniation of the stomach through the opening, and second, dilatation and paralysis of the stomach.

Local paralysis in the gastro-intestinal tract takes place when the mesentery is torn from the intestine. This phenomenon has been

observed by Moschowitz,[1] who described a case of ileus secondary to separation of the small intestine from its mesentery.

Separation of the gastric mesentery may also occur. It is usually the result of an accident. Green[2] reported its occurrence in a boy, 6 years of age, who was run over by a light ice wagon.

No previous record was found in the literature of rupture of the great omentum with herniation of the stomach occurring as a complication of whooping cough.

2014 Tulare Street.

1. Moschowitz, A. V.: Ann. Surg. **57**:262, 1913.
2. Green, N. W.: Ann. Surg. **61**:631, 1915.

ATRESIA OF THE TRICUSPID ORIFICE

GEORGE R. MURPHY, M.D.
AND
LEO F. BLEYER, M.D.
ELMIRA, N. Y.

All reports dealing with atresia of the tricuspid orifice state that it is an extremely rare anomaly. Breslich,[1] reporting one of the most recent cases, reviewed the literature and stated that altogether there had been thirteen cases reported. We have noted that he did not include the case of Cathala and Tisserand [2] reported in 1926. It may be that he did not consider that this was a true case of atresia, as they described a communication between the right auricle and the right ventricle by a narrow slit (bordered by muscular folds), without the presence of the usual interventricular defect. We recently saw a case which came to autopsy, and which we now wish to report.

REPORT OF A CASE

History.—F. W., Jr., a white boy, aged 4 months, an American, a first child, born Dec. 17, 1931, was seen by one of us (Dr. Murphy) on April 9, 1932. The father was about 50 years of age, and the mother was about 25. Pregnancy had apparently been normal, with normal delivery at term and no asphyxia or complications. The patient was referred by the family physician because of difficulty in feeding since birth and because of recurrent attacks of dyspnea and cyanosis. At the time the child was first seen he was about 4 months old, and was being fed a formula of evaporated milk, water and lactose and an intermediate feeding of oatmeal gruel, and was having five "normal" stools in twenty-four hours. The child's appetite was not satisfied, and he had a tendency to vomit curds frequently. The mother said that he had a great many curds in the stools and frequent attacks of colic. He did not sleep well and was very active. The infant weighed 7 pounds and 6 ounces (3,300 Gm.) at birth, and on April 9, he weighed 8 pounds and 13 ounces (3,950 Gm.). The mother stated that for the past three days the patient had had a slight cold in the head, nasal discharge, moderate cough and moderate fever.

Examination.—Physical examination on April 9 showed the child to be cyanotic and dyspneic, with marked emaciation and a temperature of 102.5 F. by rectum. There was marked dulness, with bronchial breathing and distant râles over the upper lobe of the right lung posteriorly. The heart appeared to be somewhat enlarged to the left; the sounds were good, and there was no murmur. The abdo-

---

From the Pediatric and Pathological Department of St. Joseph's Hospital.

1. Breslich, P.: Congenital Atresia of the Tricuspid Orifice, Tr. Chicago Path. Soc. **13**:307 (June 1) 1930.

2. Cathala, M. Jean, and Tisserand: Bull. et mém. Soc. méd. d. hôp. de Paris **75**:501 (March 19) 1926.

men was somewhat distended; the liver was palpable at the costal margin, and the spleen was one fingerbreadth below the costal margin and moderately firm. The child was referred to St. Joseph's Hospital with a diagnosis of pneumonia of the upper lobe of the right lung and possible congenital heart disease.

Admission.—On admittance to the hospital, the following laboratory data were reported: red blood cells, 4,560,000; hemoglobin, 80 per cent (Sahli); white blood cells, 10,200; polymorphonuclears, 60 per cent; lymphocytes, 36 per cent; transitionals, 4 per cent, and blood type III (Moss); both the baby's and the mother's blood were compatible. Urinalysis gave negative results, except for a reaction of 4 plus to the test for albumin and to the test for pus cells, and the presence of many hyaline and granular casts. Repeated urinalyses and blood counts gave approximately the same results. The report on the roentgen examination made on admittance stated that the chest showed general bronchitis. On April 12, a roentgenogram showed bilateral bronchopneumonia. On April 23, roentgen examination showed pneumonia of the upper lobe of the right lung. The physician in charge of the roentgen department would not commit himself as to whether or not the heart was enlarged, although it appeared to some of us that it was definitely enlarged, especially to the left. The day after admission, the child was given 50 cc. of the mother's blood intraperitoneally. His temperature while in the hospital varied between 104 F. on admission to 105.8 F. on April 12, gradually subsiding to 99.4 F. on the day of death. The respirations ranged from 20 to 80, and the pulse rate stayed around 140, although at times it increased to 180. Because of the child's marked cyanosis, it was found advisable to keep him in an oxygen tank at times. This relieved the cyanosis, but had no marked effect on the dyspnea. The child remained extraordinarily alert, and it was necessary to give him frequent doses of bromides in the hope that he would not be too active. On April 18, bilateral otitis media developed, and the drums were incised by an otologist. On

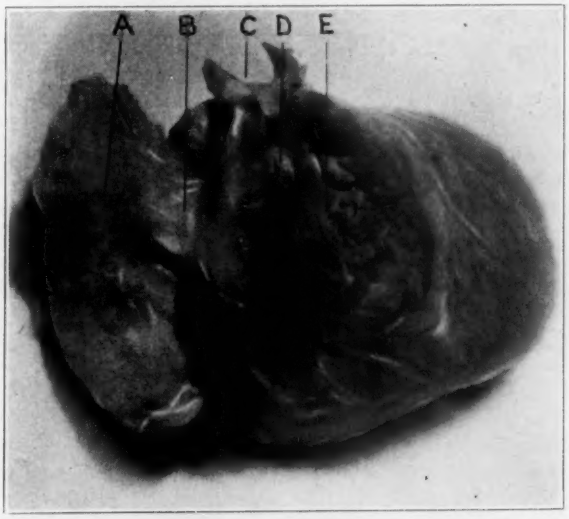


Fig. 1.—*A*, right auricle; *B*, fibrous band between right auricle and ventricle; *C*, pulmonary valve; *D*, ventricular septum defect, and *E*, rudimentary right ventricle.

the same day, the child began to have marked edema of the legs, face and cheeks, and because of this small doses of digitalis were given by mouth. The edema improved to a great extent following this, and he remained relatively free from edema thereafter. He showed very few other symptoms, except a marked distension of the abdomen, for which he was treated with turpentine stoops and enemas, and a slight irritation of the buttocks, which cleared up with a local dusting powder. When the cyanosis and edema were the most marked, the liver would vary in size to two or three fingerbreadths below the costal margin.

The patient died at 1:05 p. m. on April 25.

Autopsy.—The protocol of the postmortem observations and their correlation were reported by one of us (Dr. Bleyer). External inspection disclosed slight

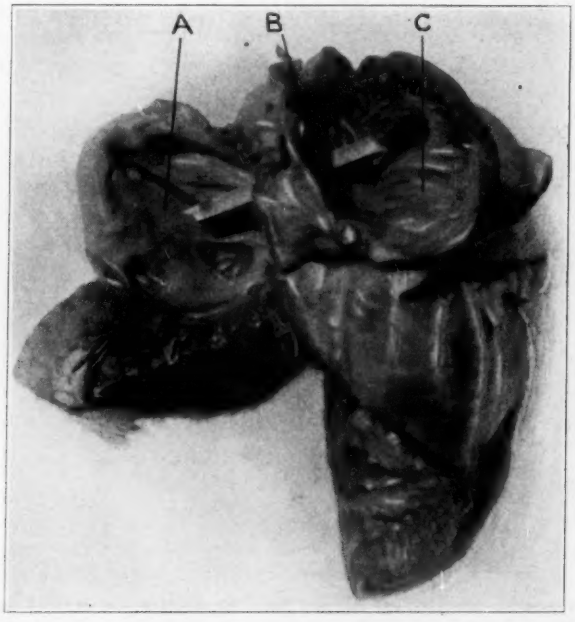


Fig. 2.—*A*, left auricle; B, open foramen ovale, and *C*, dilated right auricle.

tibial edema and a slight cyanotic discoloration of the lips and finger-nails. Internal examination was limited to the thoracic and abdominal cavity.

The pericardial sac was distended, fluctuating and filled with about 50 cc. of serohemorrhagic fluid; the inner aspect of the pericardium was smooth and glistening. The epicardium showed numerous ecchymoses. The heart weighed 28 Gm. Its configuration was abnormal, the apex being formed by the left ventricle only, and the right auricle was found to be converted into a dilated flabby pouch (3 by 2 cm.) which communicated through the patent foramen ovale with the left auricle (2 by 2 cm.). No orifice between the right auricle and the right ventricle could be made out, the former representing a blind sac; a funnel-shaped depression in the wall of the right auricle indicated where the tricuspid valve was supposed to be. A solid fibromuscular band connected the right auricle with the right ventricle.

The right ventricle was rudimentary in appearance and impressed one as a mere dilated conus of the pulmonary artery; it measured 1 cm. in length and 0.6 cm. in transverse diameter, and communicated with the left ventricle through a

small opening in the upper central part of the ventricular septum, which was covered by a continuous endocardial lining and situated just beneath the aortic cusps. The pulmonary valve was normally developed and measured 2 cm. in circumference. The left ventricle was markedly hypertrophic in appearance, its wall being firm and measuring 0.9 cm. in thickness; the musculi papillares were thickened. The length of the left ventricle from the apex to the aortic valve was 3.5 cm. and its greatest transverse diameter was 2.5 cm. Aortic and mitral valves were fully developed and showed no gross lesions. The circumference of the aorta above the insertion line of the cusps measured 3 cm.

From these anatomic changes, the circulation of the blood in the heart during life could be reconstructed as follows: The venous blood returned to the heart through the venae cavae, forced its way through the patent foramen ovale into the left auricle, this outlet, however, having been insufficient to secure complete emptying of the right auricle and having caused dilatation of it. From the left auricle, the mixed venous-arterial blood was expelled through the mitral valve into the left ventricle, where it divided into two currents. The main current passed out through the aortic valve into the arterial system, and the smaller one passed through the defect in the septum into the rudimentary right ventricle and from there through the pulmonalis into the lungs. Judging from the small capacity of the right ventricle, an insufficient amount of blood was circulated through the lungs to secure optimal oxygenation of the blood. The double function of the left ventricle, namely, to propel the blood through the arterial system as well as through the lungs, accounted for its hypertrophy.

In correlating these developmental cardiac defects, we feel, in accordance with the interpretation given by other authors, that the defects in the auricular and ventricular septums as well as the hypoplasia of the right ventricle were changes secondary to the primary tricuspid atresia. The blood stream forcing its way under pressure through the auricular and ventricular septums necessarily prevented their complete closure in embryonic life. The right ventricle failed to develop because of insufficiency of the blood supply, which normally acts as a physiologic stimulus to growth. The circumference of the pulmonalis was shorter than that of the aorta, which points to a considerable narrowing of the pulmonary artery, as it is supposed to be wider than the aorta in early childhood.

The lungs were firm and solid in consistence and were airless, with the exception of a compensatory emphysema in the marginal portions. The cut surface was dry and a uniformly dark reddish brown.

Examination of the abdominal cavity revealed changes secondary to the insufficient oxygenation of the blood. The spleen bore the alteration of marked cyanotic induration; the liver presented the signs of fatty degeneration, and the kidneys were in a state of marked passive venous congestion.

The gastro-intestinal tract, pancreas, suprarenals, urinary bladder and genital organs showed no appreciable gross abnormalities.

Microscopic examination of the solidified portions of the lungs showed the alveoli densely filled with a granulation tissue composed of fibroblasts, exfoliated alveolar epithelial cells, polymorphonuclears, round cells and a number of heart failure cells. The interacinar septums were obscured in places, and the periacinar capillaries more or less collapsed. The larger blood vessels in the stroma were poorly filled with blood. The lumens of the bronchi contained inflammatory cells and desquamated cells of the epithelial lining.

The following pathologic-anatomic diagnosis was made: Congenital atresia of the tricuspid valve, associated with hypoplasia of the right ventricle, patent foramen ovale and defect of the ventricular septum; diffuse bilateral bronchopneu-

monia with delayed resolution; cyanotic induration of the spleen; fatty degeneration of the liver, and passive congestion of the kidneys. The poor vascularization of the lungs possibly accounted for the fact that the pneumonia failed to undergo resolution as absorption of a pneumonic exudate presupposes a rich blood supply.

The widespread pneumonic infiltration was considered as the direct cause of death. It is doubtful, however, if the congenital heart lesion in itself would have been compatible with life for a longer time, as most of the patients in the previously reported cases died in early childhood.

COMMENT

The cases of this anomaly which have been previously reported have shown either recurrent or continuous cyanosis. In the case of Wieland,[3] the child had a bluish discoloration of the skin, a murmur in the last few weeks of life, no enlargement of the liver and spleen and polycythemia with 8,500,000 red blood cells. Two patients whose cases were reported by Kühne [4] showed a constant systolic murmur, with intermittent cyanosis, enlarged liver and spleen, dyspnea, indigestion and failure to gain weight, and death at the ages of 7 and 9 months, respectively. Holt and Howland [5] have stated that in 45,000 dispensary and hospital cases they found 173 cases of congenital heart disease, and considered congenital lesions of the aortic, mitral and tricuspid orifices so rare that they could be disregarded. Few of the standard textbooks on pediatrics or other books dealing with congenital cardiac malformations describe this type of case. Grulee [6] stated that Hess in 1918 was able to collect 9 cases of tricuspid atresia that were primary in origin. Cabot,[7] quoting Dr. Maude E. Abbott, found no case of tricuspid atresia in 631 cases of heart disease, although Dr. Abbott in Blumer's "Bedside Diagnosis" [8] mentioned several cases of tricuspid or mitral atresia with or without transposition of trunks. This defect, as brought out by Breslich [1] in the discussion of his case, would seem most likely to be one due to maldevelopment rather than to infection.

SUMMARY AND CONCLUSION

A case is reported with postmortem observations of an infant about 4 months old showing congenital atresia of the tricuspid orifice with

3. Wieland, Emil: Jahrb. f. Kinderh. **87**:320, 1914.

4. Kühne, Marie: Jahrb. f. Kinderh. **71**:16, 1906.

5. Holt, L. E., and Howland, J.: The Diseases of Infancy and Childhood, New York, D. Appleton and Company, 1926.

6. Grulee, Clifford G.: Clinical Pediatrics, New York, D. Appleton and Company, 1926, vol. 3, p. 109.

7. Cabot, Richard C.: Physical Diagnosis, ed. 7, New York, William Wood & Company, 1919, p. 271.

8. Abbott, Maude E., in Blumer, G.: Bedside Diagnosis, Philadelphia, W. B. Saunders Company, 1928, vol. 2.

interventricular septal defect, patent foramen ovale and rudimentary right ventricle. In addition to the cardiac condition there was a diffuse bronchopneumonic solidification with delayed resolution and beginning carnification. The most important symptom was recurrent attacks of cyanosis and dyspnea. If one includes the case of Cathala and Tisserand,[2] this case will be about the fifteenth reported showing this anomaly of the heart.

CONGENITAL ABSENCE OF GALLBLADDER AND EXTRAHEPATIC DUCTS

J. MONTGOMERY DEAVER, M.D.
PHILADELPHIA

Although a number of cases of congenital absence of the bile ducts have been reported, the condition is sufficiently rare to warrant an additional clinical and pathologic report, especially because the subject survived one year and twelve days.

Lavenson,[1] in 1908, in a review of sixty-two cases of congenital obliteration of the bile ducts, reported that only three patients had lived more than eight months; two had lived nine months, and one, eleven months. Ladd,[2] in 1928, reported twenty cases of congenital atresia with stenosis of the bile ducts, in eleven of which the patients were subjected to operation, but the condition was amenable to surgical treatment in only eight. Six of these patients recovered; in other words, 40 per cent of Ladd's twenty cases were operable. Warren Cole,[3] in 1931, reported four cases of complete absence of all extrahepatic ducts. One of these patients was operated on at the age of 7 months and was still living at the age of 8 months; the other three lived only a few months.

REPORT OF CASE

History.—J. R., a white girl, aged 8 months, was admitted on June 14, 1931, to the Children's Hospital of the Mary J. Drexel Home to the service of Dr. Charles Fife. The chief complaint was yellowness of the skin. No abnormality had been noted until the child was 6 weeks of age, when the mother noticed that the sclerae were slightly jaundiced. This condition persisted, varying in degree from day to day. The stools were clay-colored and the urine dark. Both parents were living and well. The patient was the first child. Pregnancy had been normal, and delivery had occurred at full term, no instruments being used. The weight at birth was 6½ pounds (2,953 Gm.). The patient had had a discharge from the ears for the past two months.

The infant was breast-fed for the first two months, and then it was put on a formula of cow's milk. Although the child took its feedings well and retained

---

Read before a meeting of the Philadelphia Pediatric Society, Feb. 9, 1932.

1. Lavenson, R. S.: Congenital Obliteration of the Bile Ducts with Cirrhosis of the Liver, J. M. Research **18**:61, 1908.

2. Ladd, W. E.: Congenital Atresia and Stenosis of the Bile Ducts, J. A. M. A. **91**:1082 (Oct. 13) 1928.

3. Cole, W. H.: Congenital Malformations of the Intestinal Tract and Bile Ducts in Infancy and Childhood, Arch. Surg. **23**:820 (Nov.) 1931.

them, it did not gain in weight or develop physically. There was a tendency toward constipation. Mental development was normal.

Examination.—Physical examination revealed the following essential points: The patient was an undernourished, underdeveloped white girl of 8 months; the weight was 10 pounds (4,535 Gm.); the skin and the sclerae were deeply jaundiced. The edge of the liver was palpable at 5 cm., and the spleen at 2 cm. below the costal margin. The axillary and inguinal glands, the size of peas, were definitely palpable.

Laboratory Data.—Repeated examinations of the stools and urinalyses gave negative results for bile pigment. The blood count showed a moderate secondary anemia; there were 70 per cent hemoglobin, 3,200,000 red cells and 18,000 white cells, with 42 polymorphonuclears, 47 lymphocytes, 10 per cent transitionals and 1 eosinophil. The icteric index was 60, and the fragility test gave normal results. The blood platelets numbered 136,000. The Wassermann and Kahn tests were negative. The intradermal tuberculin test was negative.

Course of Illness and Operation.—There was an afebrile course, with the condition changing little, except that the jaundice varied from day to day. Approximately one month after admission to Dr. Fife's service, the child was transferred to the surgical service with the diagnosis of congenital atresia of the bile ducts.

An exploratory laparotomy was performed under ether anesthesia. When the abdomen was opened, the liver was found to be markedly enlarged and in an advanced stage of biliary cirrhosis. The spleen was firm and large. Exhaustive exploration failed to reveal the presence of the gallbladder or any extrahepatic ducts. The foramen of Winslow was patulous. The abdomen was closed without drainage. The patient had little postoperative reaction, and the wound healed by first intention. Two months after the operation, the condition remaining unchanged, the parents were allowed to take the child home.

The patient was readmitted two months later. At this time, she had a severe cold in the head with a marked watery nasal discharge. The weight was 9½ pounds (4,308 Gm.). Urinalysis showed many pus cells. The stools were loose and coffee-ground in color, and positive for blood. The temperature was septic. The child died on October 27.

Autopsy.—Autopsy was performed by Dr. Stanley P. Reimann. The liver was freed and removed en masse with the duodenum, pancreas and stomach. The gallbladder was absent, and careful dissection failed to reveal evidence of extrahepatic ducts. Both macroscopically and microscopically, the liver showed marked biliary cirrhosis with proliferation of the intrahepatic ducts. The duodenum was normal in size and contour. The papilla of Vater was absent, and only a small opening was present for the pancreatic duct. The stomach was normal. Multiple hematogenous abscesses were found in the renal cortex and parenchyma. The spleen showed passive congestion, fibrosis of the pulp and hemosiderin pigmentation. The remaining organs, including the pancreas, were essentially normal.

CONCLUSIONS

An exploratory laparotomy was done in this instance on the diagnosis of congenital abnormality of the bile ducts or atresia of the papilla of Vater.

A definite preoperative diagnosis cannot be made between complete or partial absence or simple atresia of the bile ducts.

The condition must be differentiated from icterus neonatorum, cysts and tumors of the pancreas and hemangiomatous malformations of the liver.

Such congenital abnormalities and atresia are sometimes amenable to surgical intervention. Therefore, patients should be given the benefit of operation.

Since it is well known that obstruction of the biliary ducts leads to cirrhosis, and that chronic jaundice and intestinal disturbances create a tendency to infection, it is recommended that operation be not delayed too long.

Progress in Pediatrics

HISTORY OF INFANT FEEDING

ARTHUR BATES LYON, M.D.
BOSTON

All down through history the consensus has been that the young infant is best nourished at its own mother's breast. But in the matter of infant feeding, as indeed in many other human affairs, practice has by no means always marched with precept. Very early in history, and in many civilizations, there were mothers who sought to escape from the method laid down by nature.

In the younger days of Egypt's glory there is more reason than not to believe that maternal nursing was the rule. Egyptian culture was matriarchal in type, and the child was held in high regard. Nor is it quite without significance, perhaps, that the divine Isis is "usually represented in the act of suckling the infant Osiris";[1] a function thus practiced by a celestial mother should have been hallowed in the eyes of the daughters of earth.

But increasing wealth and perhaps the influx of foreign ideas brought a change, and Egyptian mothers of a later day very often gave over the suckling of their young to others. According to the legend of the Old Testament, a Pharaoh's daughter once found herself in possession of a handsome man child whom she had found, she explained, floating in an ark in the bulrushes. Others than myself, no doubt, have cocked an irreverent eyebrow over the lady's explanation, but history, kinder than its wont, has left her with an unblemished reputation. To go on with the story, her first act was to send for a wet nurse, and one was promptly found, the child's own mother, according to the Hebrew historians, who went on to identify the supposititious foundling with their own great lawgiver Moses. The applicability of the story is this: In Egypt, better than a thousand years before Christ, wetnursing was likely a well organized profession, for the whole transaction of securing a nurse for this storied child seems to have been carried out with the minimum of search and trouble.

Read before the Boston Medical History Club, Feb. 20, 1933.
From the Children's Medical Service of the Massachusetts General Hospital.

1. Garrison, F. H.: History of Pediatrics, in Abt, I. A.: System of Pediatrics, Philadelphia, W. B. Saunders Company, 1923, vol. 1.

As time went on wetnurses increased still further in numbers and importance. By the Ptolemaic age "Hellenized ladies seldom nursed their own children. Definite contracts were made with slave women, who stipulated to nourish the child with milk up to the third year for a definite sum and their living." [1] For the first half of the first year breast milk alone was given; afterward the infant was fed on cow's milk, of which there was a daily delivery. By the terms of the contract only "the best cow's milk" might be used, and unpunctuality in its delivery was severely punished, "though upon legal rather than upon sanitary" grounds.[1]

Of the nations which successively rose to greatness by the waters of the Tigris and Euphrates, few pediatric notions are known. Nevertheless, Babylonian civilization, like the civilization of Egypt, was matriarchal in tone, and the famous code of Hammurapi secured to women and children, if no special indulgences, at least strict justice under the law. The Babylonian goddess Ishtar, who personified the generative principle in nature, is commonly portrayed as nursing an infant whom she holds on the left arm.

In India vicarious nursing was sufficiently common, but the wetnurse had to be recruited from the same caste as that of the nursling.

The Greeks, for all their magnificent flights in philosophy, science and art, were given to the inhuman custom of exposing unwanted babies. Yet those who were preserved were, in most of the Hellenic states, kindly and even affectionately used. In Homeric days high-born mothers might nurse their own children. Thus in the twenty-second "Iliad" [2] Hecuba reminds Hector how as an infant he had received from her the "consolation of her breast," and in the eleventh "Odyssey" [3] the shade of Agamemnon recalls of Penelope how "we left her a bride new-wed when we went to the war, and a child was at her breast." So was the youthful Telemachus fed, yet his father Odysseus had himself been nursed (as he expressly says in the nineteenth "Odyssey") by the slave Eurycleia. And it must be admitted that had these traditional heroes lived at Athens in the heyday of that proud city, the son's case would have proved the distinct exception. For the most part, the Athenian matron scorned to nurse her own babe, and delegated that function to a slave or a hired woman, preferably a Spartan.

And as it had been at Athens so it was in Rome as soon as the citizens had attained to wealth and luxury. The onerous task of nursing her child was by no means to the taste of the patrician lady.

---

2. Homer: The Iliad, translated by Lang, Leaf and Myers, New York, The Macmillan Company, 1924.

3. Homer: The Odyssey, translated by Butcher and Lang, New York, The Macmillan Company, 1921.

Of all the ancient peoples, indeed, the Jews alone seem to have remained faithful to nature in the matter of feeding their young. A Jewish mother who failed to suckle her child failed nearly always of necessity and not from inclination. With this folk maternal nursing had, as a matter of fact, almost the status of a religious obligation, and those self-appointed guardians of the public conscience, the prophets, preached it over and over again as one of the primal duties. The historian is not even disposed to release the venerable Sarah from her lactescent task; she is presumed to have nursed the infant Isaac after she had turned 90. With this magnificent accomplishment in view, the Hebrew mother dared not be slack. It was her custom to put the baby to the breast even before the cord had been cut; she continued to feed him from the breast until he was 1½ or 2 years old. Precautions were always taken that this nursing period might not be cut short. Thus nursing widows were forbidden to marry again before the age for weaning lest lactation be interrupted by another pregnancy. In the case of twins, the mother nursed one and a wetnurse the other. This circumstance excepted, the latter had no calculated place in the Hebrew scheme of feeding. And how rigidly the scheme was adhered to is further suggested by the fact that the Talmud contains not a word on the subject of artificial feeding.[1]

Even among the peoples who freely employed the wetnurse, there were those who looked on her askance. It was certainly a widely held belief in antiquity that she might justly be charged with the fate of the infant at her breast. Nor did the concern stop with the mere matter of ounces and pounds accruing to the baby; even enlightened opinion considered that along with his nourishment he drew traits and characteristics from her whose breast he suckled. One of the best expositions of this belief is to be found in that curious miscellany of fact and commentary, "The Attic Nights" of Aulus Gellius.[4] The exponent is the philosopher Favorinus; the occasion, his visit of congratulation to the household of an aristocratic young Roman who has just been blessed with a son and heir. Favorinus was an ardent believer in the propriety of maternal nursing; the baby's maternal grandmother was not. His efforts to convert her are amusing, and in the ensuing conversation one may note that both she and Favorinus ignore the young father altogether. In short, it is perfectly clear who proposes to manage that household:

> When he (Favorinus) had asked how long the labour had been and how severe, and had learned that the young woman, overcome with fatigue and wakefulness, was sleeping, he began to talk at greater length and said: "I have no doubt she will suckle her son herself." But when the young woman's mother said to him that she must spare her daughter and provide nurses for the child in order that to the other pains which she had suffered in childbirth might not be

added the wearisome and difficult task of nursing, he said, "I beg of you, madam, let her be wholly and entirely the mother of her own child. For what kind of unnatural, imperfect, and half-motherhood is it to bear a child and at once send it away from her? to have nourished in her own womb with her own blood something which she could not see, and not to feed what she sees now alive, now human, now calling for a mother's care? Or do you perhaps think," said he, "that nature gave woman nipples as a kind of beauty spot, not for the purpose of nourishing their children but as an adornment of their breast? For it is for that reason (though such a thing is of course far from your thoughts) that many of these unnatural women try to dry up and check that sacred fount of the body, the nourisher of mankind, regardless of the danger of diverting and spoiling the milk, because they think it disfigures the charms of their beauty. In so doing they show the same madness as those who strive by evil devices to cause abortion of the fetus itself which they have conceived. . . . But since it is an act worthy of public detestation and general abhorrence to destroy a human being in its inception, while it is being fashioned and given life and is still in the hands of Dame Nature, how far does it differ from this to deprive a child, already perfect, already brought into the world, already a son, of its own familiar blood? . . .

"It is believed not without reason that just as the power and nature of the seed are able to form likenesses of body and mind, so the qualities and properties of milk have the same effect.

"Shall we then allow this child of ours to be infected with some dangerous condition and to draw a spirit into its mind and body from a body and mind of the worst character? . . . And there is no doubt that in forming character the disposition of the nurse and the quality of the milk play a great part." [4]

The outcome of this argument is not told, but one may hazard a guess. Instinctively one feels that the grandmother had what modern business cant styles a high sales-resistance. She had fashion on her side, and when fashion is matched against reason, how much chance does the latter stand? Nevertheless, whatever the effect or lack of effect of Favorinus' words in this instance, his general thesis was accepted by many successive generations of medical men, and one finds it quoted on occasion up to the sixteenth century.

Favorinus, it must be understood, was not a physician. The earliest medical writers who dealt at all systematically with infant hygiene and management were Greeks who practiced in the Roman Empire of the East. The first was Soranus of Ephesus, about the beginning of the second century A. D.; then came Oribasius, in the fourth century, and Paul of Aegina, in the seventh century. They are all important writers, the last named if for no more than that he transcribed the writings of his predecessors. But they were all, if one may judge, more or less opportunists, or at least pragmatists, who proceeded on the basis that vicarious nursing had come to stay, and who gave their efforts not to reform, but to attempts to hold wetnurses up to a reasonable stand-

4. Gellius, Aulus: The Attic Nights, translated by J. C. Rolfe, New York, G. P. Putnam's Sons, 1927. In Loeb Classical Library, bk. 12, vol. 2, chap. 1, p. 353.

ard. If they made no especial plea for maternal nursing, it may have been merely that they thought that such an appeal would have gone unheeded. One may speculate, but that is all.

As to the qualifications for the wetnurse, there would have been no major differences of opinion. For convenience, I quote from Oribasius: [5]

> One should select a nurse who has no disease of any sort, not too young and not too old. The youngest should be twenty-five years old, the oldest thirty-five years, and she should have a broad chest, large breast, and nipples which are not shut off and retracted. The rest of her body should not be excessively fat nor over lean. It is a great thing for the child that the nurse should have been delivered not long ago and preferably of a male child. Let her avoid things that are particularly drying, and salt things and things which are pungent and astringent, and sharp and bitter and very heating things, and those that have a particularly unpleasant odor, and strong savors and seasoning and other pungent things. She should also abstain from venery and should do work with the hands and shoulders, so she should grind and weave and walk about with the child in her arms. She should carry the infant in her arms up to the age of three or four months.[5]

The reader will observe that these remarks are not without a good measure of common sense. The prohibitions as to the nurse's diet, though not exactly specific, indicate that Oribasius realized that certain substances are transmissible by way of the milk which may bring anguish to the infantile recipient, and the injunction regarding manual work (for it will be remembered that wetnurses were universally drawn from a group habituated to labor) is of course wholly just.

Besides attempting to secure an adequate supply of milk by estimating the constitution and regulating the habits of the foster-mother, Soranus promulgated a method of testing milk which found wide acceptance and which continued to be recommended in textbooks for something like sixteen centuries:

> Whether the milk will coagulate properly is determined by the fact that if we put a drop on the finger-nail or upon a laurel leaf or other smooth surface it slowly spreads and when shaken, retains the drop-form; for if it flows at once in all directions, it is watery, but if it coheres like honey and does not change its drop-form, it is too thick. We may also make the test by adding twice as much water to a given quantity of milk; the solution will then take place only after a considerable time and the white color is retained to the last. If the solution takes place at once the milk is watery and it can be used still less if it forms a fibrous coagulum like that of serum. In such a condition it is indigestible. But if, after some time, it does not diffuse and sinks in such a manner that it remains at the bottom when water is poured upon it, it is cheesy, thick, and soluble with difficulty.[6]

5. Still, G. W.: History of Paediatrics, New York, Oxford University Press, 1931.

6. Ruhräh, John: Pediatrics of the Past, New York, Paul B. Hoeber, Inc., 1925.

With regard to the first substances given to new-born infants, there is one ancient practice of especial interest. Today, for the first day or two, nearly everyone gives a sugar solution—sugar of milk it may be—for the double purpose of holding the initial loss of weight to a minimum and of aiding in the purging of the bowel of meconium. Especially with the last-named objective in view, many of the ancients were accustomed to use honey, with or without the addition of other substances. Thus the Hindus gave no milk for the first four days of life, but only honey combined with butter and certain powdered roots. The practice was quite similar among the Hebrews, except that milk was not withheld. "Butter and honey shall he eat," chants Isaiah of the infant Immanuel, "that he may know to refuse the evil and choose the good." Nor was the custom unknown to occidental peoples, though it seems not to have been standardized among them till the days of Soranus and his fellow Ephesian Rufus. If, said Soranus, the baby seem not to be hungry during his first two days, it is best to offer nothing. If he cries, stewed honey is to be given, though not with butter, which is indigestible; goats' milk is the other ingredient of choice. For some reason not clear to posterity Soranus thought breast milk unwholesome for the first twenty days post partum, and he recommended that an older babe nurse the breast before the new-born one was put to it.

The virtual silence of the physicians of Greece and Rome on the subject of the milk of animals in infant nutrition is on the whole a little curious, for such milks, certainly long before Oribasius, must have been used rather frequently. Feeding bottles have been found which date from as early as the second century A. D. These were of clay. Glass bottles were known to the Romans by the fourth century. It is clear that pains had been expended on the maufacture of these bottles. And it seems hardly likely that so much effort would have been taken in the case of an article which lacked a ready market.

When and where sopped food—"pap" as it came to be called—first found favor as a staple of the infant dietary is another mystery I make no pretense of solving. Very likely the ancients used it to some extent; certainly the Arabians did; Avicenna mentions, I think, an electuary made of bread and milk with sugar. Yet the Arabians, to judge from such Englished fragments of their writings as I have seen, advanced the subject of infant feeding but little. On the contrary, in some directions they retarded it. Avicenna himself fathered the atrocious piece of advice that previous to offering solid or semisolid food to the luckless infant the nurse should chew it awhile in her own mouth.[6] Indeed, one must wait four or five centuries after the golden age of Islam before coming on anything constructive in the discussion of the infant dietary.

Something of a forward step seems to have been initiated by Bartholomaeus Metlinger, whose "Regiment der Jungen Kinder" was published in 1473. Incidentally, this book—with the really laudable ambition of making its matters available to midwives and nurses as well as to physicians—was the first of all pediatric texts to have been printed in the vernacular.

Now it has been charged against Metlinger that he went no further than to repeat what his predecessors had said, but this is not quite true. To be sure, he is not in the least original in describing the qualifications of the wetnurse, nor yet in making her the scapegoat for any illnesses her charge might suffer. When something went wrong with the baby's internal economy, he was quite as ready as had been his forebears to diet and dose and purge and bleed the poor nurse in the effort to straighten things out. All of which is no more than to say that he leaned heavily on tradition. But one must not forget that he lived in the fifteenth century and to do otherwise then, or for that matter long afterward, was a devilishly risky business. And, after all, he was something better than a mere psittaceous copyist. One thinks, to read between his lines, that he tried to justify the ancient practices to his reason before he again gave them voice, and his writing is occasionally enriched by the record of his own experience. Moreover, in certain matters he betrays the laudable determination to adhere to the regimen of nature. It is refreshing to find him saying (neither Soranus nor Oribasius nor Paul nor any of the Arabians said as much) that "the best wetnurse for a healthy child is its own mother." And he adds, "Only in sickness or where the milk does not come in or for other reasons the mother cannot nurse her child, one should choose a wetnurse." [6] All in all, I am ready to agree with Ruhräh that Metlinger's book has been persistently underrated by historical critics.

But I have digressed long enough. Should the reader desire to test his own critical judgment, I remark that Ruhräh has translated this old worthy *in extenso*. There is time here for only a brief extract or two on the matter of supplementary foods:

> One should know also about the gruel that is given to the baby after it has nursed; when the milk of the nurse is good and she has enough the child needs less gruel especially when the nursing agrees with it; when the milk is not good or when the wet-nurse is sick or has little milk or the baby does not thrive on the nursing one should give more gruel. . . .
>
> Further it should be known that children are to be nourished on gruel and milk until they cut their front teeth. One may then give somewhat stronger food, bread soaked in milk or meat soup or pea soup, and when they become older one can give them lean well-cooked meat cut very fine but in small quantity, as too much and too strong meat causes worms to grow. The normal time for a child to nurse is two years but during this time it can be allowed

other things to drink besides mother's milk, and other foods such a gruels. One may also give at this time water to drink, or water that has been once brought to a boil and then allowed to cool, or at times a well watered wine.[6]

Metlinger seems not to have been at all concerned with the matter of regularity in feeding. "The child should not be awakened for either food or drink," he insists, "as the more it sleeps the better it is for it." His sole admonition as to the feeding time is that the baby should not be fed within an hour of his bath. Concerning weaning he remarks:

. . . if the nursing agrees with the child it can be allowed to nurse but if it is not gaining in weight or if the wet-nurse becomes pregnant . . . it may be necessary to wean the child, but if the child is weaned it should be done in the proper manner so that each day the nursing is discontinued and water given to drink which has been once cooked and allowed to cool, or sugar water made with an ounce of sugar and a measure of water and boiled as long as it will take to cook a hard egg. A bread rind may be cooked in it in order that the child may be strengthened. But if the child is young, that is to say under a year, and it must be weaned, one gives it goat's milk to drink out of a cornucopia or a little jug. The milk is boiled with a quarter of water and an electuary made out of white bread and sugar, as Avicenna tells us.[6]

A little more than three fourths of a century after Metlinger, there was published another book on pediatrics which, for the range of its matters and the judgment with which they were considered, seems clearly to have surpassed anything that had gone before. This was the "Cinq Livres, De la maniere de nourir et gouverner les enfans des leur naissance," written by Simon de Vallembert and published at Poictiers in 1565. Vallembert has found of late a most enthusiastic advocate in Dr. Still,[5] who thinks that in respect to sound common sense this old Frenchman was "far in advance of most of the writers of the sixteenth century." In particular, Still commends his chapter on the feeding of the infant up to the time of weaning.

Unfortunately I cannot tell anything about the book except what Still [5] and Ruhräh [6] say, for the original is out of reach, and I know of no translation. But it appears that in regard to infant feeding Vallembert was less a slave to tradition than might have been expected—that he had, as Still insists, a mind of his own. And so, while he carried the nursing period to the age of 18 months or 2 years, as had been done from time immemorial, he was really astonishingly liberal in the matter of supplementary foods. Cow's milk or goat's milk he allowed quite early; in this milk might be boiled flour or crumbled white bread or semolina, and sometimes yolk of egg. His panade, compounded of bread crumbs, bouillon, milk and vegetables, was quite the tastiest dish yet devised for the titillation of the infantile palate. He denounced the nasty practice of prechewing the baby's food, and he allowed something in the way of solids as soon as the first teeth had been cut.

The diet prescribed by Vallembert, as has been said, was better than any which had gone before, and, it may be added, many which came after. But the modern reader, whose own dietary calculations embrace perforce such things as calories, vitamins and mineral salts, can hardly help wondering whether even a relatively good food list like Vallembert's must not have been gravely deficient in certain respects. I can say only that if this scheme raises his doubts, those now to be discussed may well horrify him.

For the English feeding schemes of the seventeenth century, and even those of the early eighteenth century, were inferior in theory, and infinitely inferior in practice, to that of the sixteenth century Frenchman. Somebody once said that the victories of England were won on the cricket fields of Eton. Perhaps it is true; at least the evolution from cricketer to conqueror is not incomprehensible. What is really difficult to understand, after surveying the infant dietetics of those days, is how young England ever lived long enough to get to Dame school, let alone Eton.

Bad as they were, however, these infant dietaries have a particular historical interest for American readers. For England was our mother in medicine as she was in language, literature, manners and political institutions. And although cases might differ somewhat in the two countries, American colonial medicine was in principle essentially a replica of medicine in the old country. Excepting possibly on our frontiers babies were fed here much as they were over there. But on that point, as on many others, the English record is better documented than ours, and is in consequence a good deal easier to follow.

There was in England, says David Forsyth,[7] on whose valuable "History of Infant Feeding Since Elizabethan Times" I shall lean somewhat heavily hereafter, no mention of artificial feeding in substitution for the breast before the eighteenth century, and hand feeding as a reasonably reliable method of infant nurture exists only from the middle of the nineteenth century. And possibly that is putting it too early.

In Tudor times feeding bottles were unknown in England; artificial foods were limited to sopped bread and pulse, while breast feeding, elsewhere stopped at the age of two, was not rarely carried on for three years. Milk from animals was not employed. This was not from lack of cattle, for even at London pastures extended to the city walls. The trouble lay in the inevitable contamination of the milk. Man and beast and their respective abodes were all dirty. At every corner of dark and narrow streets stood a heap of stinking refuse. There were latrines but no drains. Behind each house lay an open cesspool. Nor was there a chance for the overcrowded city to grow

into freer and cleaner suburbs, for the church owned all the ground about the walls.

The filth within the houses matched that without. Under the rushes which served to carpet the clay floors lay the accumulations of years: "spittle, beer, decayed fish, and other refuse even more disgusting."[7] Personal cleanliness was unknown. Even the few weeks old baby must have shared in the general habit; his small person—not to shame his progenitor's—doubtless soon came to harbor a numerous and lively entomologic collection.

It is hardly to be imagined that folk so little fastidious about themselves should have been at pains about the cleanliness of their cattle, but had the milk been ever so wholesome at milking it must have been spoiled under the prevalent conditions of preparation and storage. The one procedure which might conceivably have made it safe to use, namely, boiling, was decried even by the faculty of medicine. Nor has this Elizabethan prejudice even now quite disappeared from history.

Under the Stuarts it is possible that Englishmen possessed a few comforts hitherto unknown, but a clean body cleanly housed was certainly not among them. Indeed, on reflection it seems sure that cleanliness is far from an inherent condition in man; it is an acquired luxury that among a few peoples, and only since yesterday in history, has begun to be habituated to the seeming status of a necessity.

So in seventeenth century England babies were as dirty as earlier babies had been, and they were fed in the same old way. The little pediatric text of one John Pechey (1697) could well serve as a mirror of sorts for current practice.[8] Probably there is nothing whatever original in the book; it recommends (like all the rest) breast feeding up to the age of 2, while of artificial feeding it has hardly a word to say.

Yet this book was but warm from the press when artificial feeding—hand feeding, as our fathers called it— began to have its first extensive trial in England. This innovation originated not with the medical profession, however, but with the laity. And as a matter of fact, had some pediatric Moses arisen in the land it may well be doubted how far his authority would have served. For Georgian England held stubbornly to the notion that the fittest doctor for a baby was an old woman, an opinion which the faculty of medicine was at no pains to dispel. Perhaps indeed the medical profession even fostered the belief. At any rate, one's physician stood very much on his dignity in those days, and the suspicion will not down that he considered the ordinary everyday feeding of a puling infant quite beneath his august notice. So the eighteenth century was pretty well along before anything like an infant

7. Forsyth, D.: Proc. Roy. Soc. Med. (Sect. Dis. Child.) **4**:113, 1910-1911.
8. Garrison.[1] Still.[5] Forsyth.[7]

welfare movement was established, and not till 1797 was "the first faint light of intelligent scientific inquiry turned in the direction of infant feeding." [7] That was long after the Gampian experiments next to be mentioned—experiments which, as has just been remarked, were carried on independently of professional medicine.

As it turned out they were very costly experiments indeed. Not that a high infantile death rate was exceptional, for people were used to that. In so-called normal times two thirds of all deaths were of children under 2 years of age; in some years, and while maternal nursing was still the rule, death claimed a good half of the babies born.[9] Appalling as these figures seem to us, worse were to follow, and the feeding schemes of the early eighteenth century were almost certainly responsible.

It must be recalled that by this time England had become a wealthy nation, and its well-to-do folk (as well-to-do folk always have done) were demanding and getting more luxuries. And among these luxuries the wetnurse was coming into an increasingly important place. It was merely another case of history repeating itself, and when one considers that the conventional nursing period was still two years, the repetition is not to be wondered at. It was an onerous prospect—two years of nursing! Wealth provided the chance to escape, and fashion dictated the method. All at once wetnurses were in great demand; presently demand exceeded supply (or at least a suitable supply); and the lactant sisterhood came into all the advantages of a seller's market. Its ranks were swelled by the invasion of the immoral and the unfit. But the employer, if she was to have a wetnurse at all, could not be too finicky. She took whom she could get, often enough some lazy, ill mannered slattern, of whom (saving the resumption of her maternal job) she would have given anything to be rid. One fancies it was discomfort of this sort, repeated in a thousand homes, which led to a concerted effort to make shorter the period of breast feeding.

This could not be done, of course, unless adequate substitute foods were forthcoming. So cow's milk received its first real trial; from this time forward, indeed, it was never wholly abandoned as an infant food, but conditions were still too bad for its really successful use. For one thing, there lacked a suitable device for its administration; for another, being regularly and richly impregnated with muck and dung, it made babies sick. It was for such reasons that milk came to occupy a subordinate place in the infant dietary, and that water-pap—bread and water boiled together—took first rank among the accessory or substitute foods. The cutting of the first incisors was the signal for the exhibition of this appetizing compound, and when the next teeth made

9. Garrison.[1] Forsyth.[7]

their appearance the diet was liberalized by the addition of weak tea and broth and the minced wings of boiled chickens. Still later came bread panada sweetened with Lisbon sugar, French bread, Uxbridge rolls and the minced flesh of pigeons, larks, thrushes and ortolans. From its exotic constituents this diet sounds almost romantic in the telling, but, besides being very badly balanced, it was probably far from that in the eating!

There is reason to believe that a baby's diet in those days was as wretchedly prepared as it was abominably served. Had the ingredients been nectar and ambrosia, they would have been ruined before the infant got them. To get him fed in the easiest possible way seems to have been almost the sole aim. Pap was usually administered from a utensil resembling a gravy-boat, except for a spout which was thrust into the baby's mouth. It is conceivable that the contents of this receptacle occasionally reached the infant stomach uncontaminated. But the chances were always against it, since the prevalent practice was for the nurse to test the temperature of the mess by first passing it through her own mouth. The aforementioned Pechey (so Garrison tells [1] us) knew no better than to repeat Avicenna's monstrous advice relative to the prechewing of the baby's meat.

These, however, were not the sole factors which served to augment infant mortality. Of the literally thousands of wetnurses employed in Georgian England, many, perhaps most, were unable, either for natural reasons or because employers objected, to nurse their children along with those for whom they were hired. The unfortunate young of these women were unceremoniously dumped into almshouses, asylums or other institutions, where most of them soon died. But there were always others coming along to take the vacant places. The mortality, as Sir Hans Sloane remarked, was a good three times as great among the dry-nursed as among the breast-fed, and "death from want of breast milk" was among the commonest of entries on the hospital records of the day.[1] Some idea of the figures may be gathered from the statement that when, as a sort of forlorn hope, the British Lying-In Hospital made breast feeding compulsory, the intramural death rate among babies dropped to a mere 60 per cent!

On the situation at large, the conditions of the health and fitness among wetnurses cannot have failed to exert a considerable influence. That numbers of them were physically and morally inferior has already been shown—and why. There were more jobs than candidates; the pay was good; the conditions were easy. It is altogether likely that sundry young women, observing these facts, were quite willing to assume motherhood without benefit of book and ring simply to qualify for what looked like a pleasant sinecure. Certainly the inability to produce marriage lines was no bar to employment. Indeed some

employers, as Forsyth [7] hints, regarded the lack of a husband, who might come lolling about to distract his wife, or who might even succeed in luring her back to his own vine and fig tree, as a distinct blessing. It is a point of some importance that not all these frail sisters could have kept free from disease, and though there are no figures to tell the story, one wonders how often the pox was transmitted by them to their helpless, innocent charges.

Such in general were the principal factors underlying the appalling infant mortality of the early eighteenth century. And before the situation could be materially improved wetnursing as an organized profession had to be eliminated and a way discovered whereby cow's milk might be used with a reasonable prospect of safety. Toward the latter end the development of a suitable feeding device was urgently required.

Forsyth says that a prepared cow's horn was the first piece of nursing apparatus to be tried out in England. The feeding horn might have held a gill and a half; its tip was perforated, and over the hole were attached two small pieces of parchment or leather "shaped like a glove and sewed together in such a manner that the food passed into the horn can be sucked through between the stitches." [7] The arrangement had inherent drawbacks; by no possibility could it be really cleaned, and one reads without surprise that the small sucker of its leathern teat (how rich a culture ground it must have been) was presently wont to be seized with "watery gripes."

The "bubby-pot" (delicious name) introduced by Hugh Smith in 1770, was rightly regarded as a great improvement, and it continued to enjoy the public esteem for about a fourth of a century. Dr. Still [5] thought that Smith's own description of it was worth quoting and so do I.

> I have contrived a milk pot for my own nursery . . . it appears to my family and to many of my patients preferable to those now in use and may probably be still further improved. . . . This pot is somewhat in form like an urn, it contains a little more than a quarter of a pint; its handle and spout or neck are not unlike those of a coffee-pot except that the neck of this arises from the very bottom of the pot and is very small. . . . The end of the spout is a little raised and forms a roundish knob somewhat in appearance like a small heart; this is perforated by three or four small holes; a piece of rag is tied loosely over it which serves the child to play with instead of the nipple, and through which, by the infant's sucking, the milk is constantly strained. The child is equally satisfied with it as with the breast; it never wets him in the least; he is obliged to labour for every drop he receives; . . . and greatly in recommendation of this contrivance the nurses confess it is more convenient than a boat, and that it saves a deal of trouble in the feeding of an infant; which is the greatest security to the parents that servants will use it when they themselves are not present.

To sketch the further evolution of the feeding bottle through its several phases is beyond my purpose. Until the final stage, however,

the greatest difficulty always lay in securing a satisfactory nipple. All the earlier nipples, whether of leather or rag or, as was sometimes used, the prepared teat of a heifer, shared the defect—it was not the only one, but the one which struck the users most forcibly—of collapsing when suction was applied. This led to the practice of inserting a bit of sponge within the nipple. They did not dream, of course, that in eliminating a minor annoyance they were augmenting a major hazard, for every drop the baby drew was of necessity freshly filtered through a bacterial paradise. The situation was not to be fully remedied until the days of the glass bottle with its short, relatively incollapsible and easily cleansed nipple of rubber, inventions of the not very early nineteenth century.

By this time every textbook discussed what ought to go into the bottle; that is to say, the medical profession was at last fully awake to its duty of supervising the infant's dietary.

The chief awakener, at least in point of time, seems to have been Dr. Michael Underwood of London. Underwood was not one of the great men of medicine, but there is not much doubt that he was *facile princeps* among the pediatricians of his day, as indeed he might still have been had the competition been keener than it was. His "Treatise on the Diseases of Children," [10] published in 1797, was a monument of sagacity as measured by the standards of that time, and that it served a definite need there can be no doubt. It passed through a number of editions both at home and in America. Incidentally, it contained the first clearly defined picture of infantile paralysis. Its most currently useful chapters were undoubtedly those on infant feeding.

For the infant Underwood much preferred breast milk, but he saw clearly enough that artificial feeding had come to stay and bestirred himself concerning ways and means to make it successful. Of artificial foods he declared animal milk to be the most proper and wondered "how the custom of stuffing newborn infants with bread could be so universal." He quoted a table in which was set forth with essential accuracy the likenesses and unlikenesses of the milk of various domestic animals to human milk. Cow's milk, everywhere easily available, he thought suitable for the average well baby, but suggested asses' milk for the very delicate infant or for the infant in whom cow's milk caused purgation. In the face of popular opinion, he declared for the boiling of milk.

On the frequency of feedings and on the amounts and dilutions of the milk to be given, he was less precise. But he used barley water as a diluent, and he enriched the baby's dietary by the addition of

10. Underwood, M.: A Treatise on the Diseases of Children, ed. 2, Boston, D. West, 1806.

such starches as tapioca, semolina and rice. He was definitely opposed to the older types of feeding devices, but he singled out Hugh Smith's bubby-pot for approval, since it could be (and, as he insisted, must be) cleaned between feedings. If an infant were so fortunate as to be breast fed, 1 year was assigned as the age for weaning.

I do not mean to carry these random notes much further, for to cross the threshold of modern times would take a great deal too long. I can indicate only in the briefest fashion the developments between Underwood and the present era.

The three major developments of the nineteenth century were, according to Forsyth,[7] the general acceptance of the principles of artificial feeding, the production of special foods for babies (since become an enormous industry) and the perfection of a satisfactory nursing bottle.

The first proprietary infant foods were forthcoming about 1840, but while they admittedly increased the armamentarium of the pediatrician, they by no means brought the answer to all his problems. They did not displace cow's milk; they were merely added to it, and if the milk was inferior they could do nothing to redeem the situation.

The milks of a generation or two ago were still far from being above reproach. I am not sure that all of them are even now. Nevertheless, the modern city dweller has certain unexceptionable supplies at his command, for health authorities and even the general public are now awake to the conditions of milk production, and this, together with quick handling, continuous refrigeration and, above all, the widespread use of pasteurization, has brought a beneficent change. Under current conditions it is possible for cattle to be pastured hundreds of miles from the consumers of their milk, and for that milk to remain safe for any use. But it could not be done until the health of both the cattle and their handlers was carefully guarded; it could not be done when transportation was of necessity slow, nor until continuous cold storage could be provided.

Historically speaking, the facilities which one takes so much for granted are among the newest of things. Had one visited London about the middle of the last century, one would have found that since milk could not be brought from any distance into the city the cattle themselves were housed there, in cellars "dark, unventilated, and filthy with the wretched animals, full of disease, and crowded almost to suffocation."[7] Nor could one have prided oneself that things were better in America. LaFetra[11] relates that in New York in 1842

11. LaFetra, L.: Arch. Pediat. 49:36 (Jan.) 1932.

> . . . more than half the milk used in the city came from cows fed on distillers' mash. At 9th Avenue and 18th Street, adjoining a distillery, were sheds containing 2,000 cows. The stables were rented at five dollars a year per cow, and mash supplied at nine cents a barrel. City tramps were the milkers, being paid for their labor by night shelter in the stables. The cows were diseased and the milk filthy.

It was only with the rise of bacteriology a few decades later that men began to realize the microscopic horrors which might lurk in milk, and an ever lengthening series of experiments served to pile proof on proof that this innocent-appearing fluid might carry—and did carry with alarming frequency—the germs of death and destruction. The truth was not to be suppressed, and despite the opposition of the ignorant and the mercenary, the cleansing of the Augean stables—in this case the cow-barns of a nation—was presently begun. It was a slow task and a hard one; it is not even yet complete, but no longer does one look for an outbreak of acute diseases along the trail of the milk wagon.

No, the world has become safer for babies. Hand feeding has been robbed of its former terrors; a workable knowledge exists concerning the total and fractional food requirements of the infant; the necessity for, and the functions of, sundry vitamins and mineral salts are to a considerable degree understood, and to meet the several requirements a vast array of foodstuffs is offered for one's selection. Yet with all these substances at command, with the factor of safety in artificial feeding greater by far than ever before, the lesson still comes home to the medical profession that no method of feeding ever devised, nor any probably that ever will be, can compare with that laid down by Dame Nature.

ATROPHIC CIRRHOSIS IN CHILDREN

V. H. MOON, M.D.
PHILADELPHIA

The term atrophic cirrhosis, as used in recent literature, is synonymous with portal, Laënnec's, nodular, granular and alcoholic cirrhosis. The term refers to this form of hepatic disease. It does not imply decreased size of the liver. In about 40 per cent of cases of this type, the weight of the liver is above normal. The liver is regularly enlarged in the earlier stages of this form of cirrhosis, and in children it usually remains above normal size. Confusion arises when authors class these large livers as hypertrophic. Mallory avoids the paradox of classifying large cirrhotic livers as atrophic by using the term alcoholic cirrhosis. He states emphatically that alcohol is not the cause of such cirrhosis. Thus, in avoiding one source of confusion another confusing term is perpetrated. All this confusion of terms might be avoided by the use of another of the synonyms for the atrophic type of cirrhosis.

Atrophic cirrhosis in its typical form is a well defined entity both in its clinical manifestations and in its characteristic pathologic features. From a summary of 320 cases occurring in children, Seitz [1] gives the manifestations which may be expected. Its onset is gradual and is evidenced by loss of appetite, malaise, digestive disturbances and increased size of the abdomen. A subicteric pallor is regular; moderate icterus is present in a majority of cases, and the icterus occasionally is profound. The urine usually decreases in amount and becomes dark. Slight anemia develops gradually into marked anemia. Red cell counts between two and three million and hemoglobin of from 40 to 50 per cent are usual. In a majority of cases there is fever. Some patients have a subnormal temperature. Frequently there is leukopenia but the white cells may be normal or increased in number. Both the liver and the spleen become markedly enlarged; later the liver usually shrinks somewhat and becomes hard. A tendency to bleed may be present throughout the course. The later stages of the condition are marked by increased tendency to bleed, prominence of superficial abdominal veins,

---

From the Department of Pathology, Jefferson Medical College.

1. Seitz, C., in Pfaundler and Schlossman: Handbuch der Kinderheilkunde, ed. 3, Berlin, Julius Springer, 1924, vol. 3, p. 359.

ascites, cachexia and frequently by pleural effusion. Often the terminal complication is pneumonia, peritonitis, pleuritis, pericarditis or hemorrhage.

The features just described should be compared, item by item, with those of Banti's disease. Cirrhosis in children runs a more rapid and acute course than in adults. In many of the reported cases the duration was less than two years. In a case which I studied,[2] death occurred within ten weeks from the beginning of symptoms. In such cases the clinical features of Banti's syndrome are not so characteristic as in those in which the course is more gradual, lasting two or more years after the onset.

The essential pathologic condition is found in the liver and spleen. Both of these organs are regularly involved. The liver may be larger or smaller than normal but is firm and irregularly nodular and cuts with increased resistance. Microscopic examination shows a chronic proliferative inflammatory process. Bands of fibrous tissue surrounding nodules of liver cells, moderate or marked chronic inflammatory infiltration, total obliteration of the normal lobular architecture and proliferation of bile ducts constitute the microscopic picture. The spleen is greatly enlarged and firm. Its weight may almost equal that of the liver, as in 2 cases which I studied. In a girl, aged 12, the spleen and liver weighed 930 and 980 Gm., respectively, and in a boy aged 13, 1,120 and 1,330 Gm., respectively. The capsule is thickened and usually is adherent to adjacent structures. On section, the splenic substance is seen to contain little blood, and it has a beefy firmness and color. Microscopically, the sinuses are widened and prominent. There is a diffuse increase in fibrous elements and a diminution of the cell of the splenic pulp. Varying amounts of hemosiderin deposit are present. The splenic follicles are fewer and show varying degrees of atrophy and replacement by fibrous tissue.

Hemorrhages, ascites, edema, dilatation of abdominal veins, pleural effusions and terminal infections, such as pneumonia, complete the pathologic picture.

Recent reports of cases create the impression that atrophic cirrhosis occurs rarely in children. Sutton[3] reported what he believed to be the thirteenth case of atrophic cirrhosis of nonalcoholic origin on record in America. He found records of 10 cases in children who at some time had taken alcohol. Calvin and Saffro[4] summarized the reports

2. Moon, V. H.: Infection as a Cause of Juvenile Cirrhosis, Am. J. M. Sc. **177**:681, 1929.

3. Sutton, T. L.: Cirrhosis of the Liver in Childhood, Am. J. Dis. Child. **39**:141 (Jan.) 1930.

4. Calvin, J. K., and Saffro, L. B.: Cirrhosis of the Liver in Children, Am. J. Dis. Child. **43**:914 (April) 1932.

of 23 cases referred to by Sutton and added a report of a case. Bridgeman and Robertson [5] reported what they believed to be the fourteenth case of atrophic cirrhosis in children recorded in the American literature.

Inaccuracy may result from overemphasis on the rarity of atrophic cirrhosis occurring in children. The small number of reported cases results in part from the fact that many pathologists do not regard cirrhosis in children as sufficiently rare to justify report. Within three years, 3 patients were brought to Jefferson Hospital. In each case, the cirrhosis was atrophic and ended fatally. Studies were made on the possible cause of juvenile cirrhosis,[2] but these were not placed in the literature in the form of case reports. Later, I was seeking additional cases for study on the histogenesis of atrophic cirrhosis.[6] My colleagues furnished specimens from 4 cases, none of which they had taken the trouble to report. Hanau [7] saw 4 cases in one year. Seitz summarized 320 reports of cases. He found 31 among infants, 72 among young children and 217 among older children. The United States Mortality Statistics for 1929 show 64 deaths from cirrhosis among children under 15. In ten years, from 1920 to 1929, there were 832 such deaths.

The number of cases shown by mortality statistics is below rather than above the actual number as shown by postmortem records. Thus in the ten years from 1920 to 1929, the total occurrence of cirrhosis as a cause for death, according to the United States Mortality Statistics, was less than 1 per cent. In 4,507 postmortem examinations, Mallory, Parker and Nye [8] found 224 cases of cirrhosis, or 5.08 per cent. Ophüls [9] found 166 cases of cirrhosis in 3,000 postmortem examinations, or 5.5 per cent. These figures, of course, include all ages. It may be assumed with safety that its occurrence in children as shown by mortality statistics is below the actual number revealed by postmortem examinations.

The relative frequency of cirrhosis at different ages is shown in the following figures from table 7, United States Mortality Statistics, 1929: under 1 year, 14; 1 year, 3; 2 years, 6; 3 years, 4; 4 years, 4; from 5 to 9 years, 14; from 10 to 14 years, 19, and from 15 to 19 years, 29.

---

5. Bridgeman, M. L., and Robertson, T. D.: Familial Juvenile Cirrhosis of the Liver, Am. J. Dis. Child. **43**:1155 (May) 1932.

6. Moon, V. H.: Histogenesis of Atrophic Cirrhosis, Arch. Path. **13**:691 (May) 1932.

7. Hanau, F.: Beitrag zur Kenntnis der hepato-lienalen Systemerkrankungen, Monatschr. f. Kinderh. **29**:54, 1924.

8. Mallory, F. B.; Parker, T., and Nye, R. N.: Experimental Pigment Cirrhosis Due to Copper, J. M. Research **42**:461, 1921.

9. Ophüls, W.: A Statistical Survey of 3,000 Autopsies, Stanford University Publication, Series 1, M. Sc., 1926, p. 131.

Exact figures on the occurrence of different types of cirrhosis in children are not available. More than 90 per cent of adults who die of cirrhosis show the atrophic form at postmortem examination. In the majority of the patients under 1 year of age, the condition is obstructive biliary cirrhosis which, being due to congenital conditions, usually ends fatally within a year. The vast majority of cases of cirrhosis occurring after the first year are of the atrophic type, otherwise known as portal or Laënnec's cirrhosis.

In 1929, cirrhosis caused 1 death for each 3,845 deaths from all causes among children under 15. It occurs more frequently in males than in females in a ratio of 8: 5, which ratio is identical to that found in adults.

That the liver and spleen form a sort of unitary system, both members of which are affected by the same disease, is a view advanced chiefly by European writers.[10] It is observed that such diseases as usually affect both members of the hepatosplenic system may occasionally affect one member alone, leaving the other organ normal or with relatively slight involvement. The relation between hepatic and splenic involvement in cirrhosis affords perhaps the best illustration. In the great majority, over 90 per cent of cases of atrophic cirrhosis, the spleen is involved. Splenic enlargement and a mild but progressive anemia usually precede any evidence of hepatic disease. Banti noted this and he interpreted the accompanying manifestations as a separate disease entity. Those manifestations constitute the characteristic early course of cirrhosis, when that course, as in children and young adults, is sufficiently acute to be recognized. In adults, the onset may be so gradual and insidious that symptoms of either splenic or hepatic disease are not shown. At postmortem examination in such cases the splenic involvement is regularly present. Banti's disease presents no clinical features by which it can be differentiated from those which are characteristic of atrophic cirrhosis in young persons. At postmortem examination of patients presenting the clinical syndrome of Banti's disease, the observations are indistinguishable from those of atrophic cirrhosis in patients of similar age. The authors cited and other writers both in Europe and America do not recognize Banti's disease as an entity either clinically or pathologically. They admit its existence only as a symptom complex frequently presented by a combined involvement of the spleen and liver—hepatosplenic disease, as seen in cirrhosis.

Unusual cases occur in which the disease is almost or entirely limited to one or the other of these two organs. The cases of splenic anemia without cirrhosis described by Osler may be cited as illustrative. This

10. Eppinger, H.: Die hepato-lienalen Erkrankungen, Berlin, Julius Springer, 1920. Hanau.[7]

condition presents some of the clinical features of Banti's syndrome but is lacking in associated hepatic manifestations. The spleen presents gross and microscopic changes identical to those found in Banti's syndrome or in cirrhosis. Conversely, occasional cases of atrophic cirrhosis are seen in which involvement of the spleen is slight. Such cases are unusual. The evidence for the conception of hepatosplenic disease is supported by the fact that frequently the course may be interrupted by surgical removal of the spleen. It would appear in such cases that the involvement is primarily splenic, and that splenectomy removed the chief cause before the disease progressed to irreparable damage of the liver. Pearce, Krumbhaar and Frazier [11] adopt a similar conclusion on the causative relationship of the spleen in such cases.

The conception of hepatosplenic disease as set forth coincides with much of the clinical and pathologic evidence concerning cirrhosis, Banti's syndrome and splenic anemia. It merits thoughtful consideration.

Rössle [12] made an exhaustive review of the literature bearing on the pathology of cirrhosis. He found no evidence of a hereditary familial tendency. Instances of its occurrence in successive generations in the same family are exceptional. The reverse is true of its familial occurrence in the same generation. When cirrhosis occurs in childhood, it not infrequently affects more than one among brothers and sisters. Rössle's review does not substantiate either Banti's disease or Hanot's cirrhosis as clinical or pathologic entities. In instances of familial occurrence there is frequently a history of acute infection preceding the onset. As early as 1887, Howard [13] reported cirrhosis in a sister and brother, aged 9 and 8 years, respectively, in which he suggested infection as a probable cause. In each, the disease ran a markedly febrile course following pertussis. Ely [14] reported cirrhosis occurring in twins at the age of 4. The disease began with jaundice, which developed during measles and persisted until death. The course was marked by intermittent fever. Three cases reported by me [2] occurred in a family of children with a history of scarlet fever. Hemolytic streptococci were obtained in large numbers and in pure culture from the liver in one of these cases at postmortem examination. Abt [15]

11. Pearce, R. M.; Krumbhaar, E. B., and Frazier, C. H.: The Spleen and Anemia, Philadelphia, J. B. Lippincott Company, 1918, p. 253.

12. Rössle, R.: Entzündungen der Leber, in Henke and Lubarsch: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1930, vol. 5, p. 274.

13. Howard, R. P.: Cirrhosis in Children, Am. J. M. Sc. **94**:350, 1887.

14. Ely, T.: Familial Occurrence of Cirrhosis, Boston M. & S. J. **170**:542, 1914.

15. Abt, I. A.: Hanot's Cirrhosis in a Two Year Old Child, M. Clin. North America **3**:13, 1919.

suggests infection as a cause for cirrhosis. Banti maintained that the syndrome he described was of infectious origin, notwithstanding unsuccessful attempts to isolate the infectious agent. In his review of 320 cases, Seitz attributed about 33 per cent of them to infection.

SUMMARY

The atrophic type of cirrhosis occurs in children more frequently than isolated reports of cases might indicate.

The course of the disease is shorter and its manifestations more acute than in adults.

Frequently the clinical and pathologic features are not distinguishable from those of Banti's syndrome.

The regularity with which splenic involvement accompanies cirrhosis supports the conception of hepatosplenic disease.

The author believes that cirrhosis both in children and in adults results from a combination of factors rather than from a single cause, and that infection is one important factor.

Pediatric Biographies

JAMES NELSON
1710–1794

JOHN RUHRÄH, M.D.
BALTIMORE

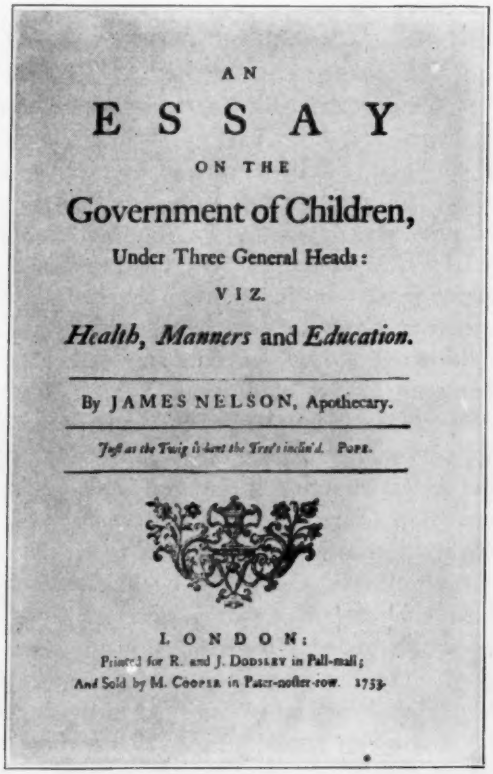
Red Lion Square, Holborn, London, has, as Caulfield pointed out, a peculiar interest for pediatricians, for it was here that Armstrong started his first dispensary, here lived Joseph Hanway, one of the great exponents of infant welfare of the eighteenth century, here "dy'd Dr. Harris, a physician," and here, too, for fifty years lived James Nelson, apothecary. Apart from the fact that he was born in 1710 and died in 1794, and that between these dates he practiced pharmacy for many years, we know little, but Nelson was interested in children and their welfare and was what today would be called an "uplifter," for he wrote "The Affectionate Father, a Sentimental Comedy, Together with Essays on Various Subjects," which appeared in London, in 1786. It is a play intended to inculcate moral truths. The *Monthly Review,* in commenting on this play, said: "Mr. Nelson's scenes aim at the noblest end, and in a moral sense are superior to the wit and brilliancy of a Congreve." All of which goes to show that going in for morality does not always or even often tend to perpetuate one's name. At the time of Nelson's death, the *Gentleman's Magazine* said of him: "The eminent virtues of his heart had long endeared him to a numerous acquaintance. He had resided in the same spot upwards of half a century. Peculiar cheerfulness and benevolence of heart marked the whole of his long and useful life; in the cause of which he experienced many and severe trials; all of which he bore with Christian fortitude and resignation."

"An Essay on the Government of Children, under Three General Heads: viz. Health, Manners and Education," was about the only thing which James Nelson did to achieve fame. It was published in London by R. and J. Dodsley, in Pall-Mall, in 1753; a second edition appeared in 1756, and a third in 1763.

If the author leaned on John Locke, the physician-philosopher, he had good support. The second part on manners deals with the training of children and the third is on schooling and older children. The first part on health is of chief interest. One gets some idea of the different customs prevailing in his day, but human nature has not changed. Some of his ideas and quotations he derived from William Cadogan, another

sound source. Most of his opinions would be regarded as orthodox today, when health is accounted more important than religion, at least by the majority. A few examples culled at random will show his style and opinions, and one or two which differ a bit from the present ideas are also given.

First then, I earnestly recommend to both Father and Mother, that their Children suck the Mother's breast.



AN

ESSAY

ON THE

Government of Children,

Under Three General Heads:

VIZ.

Health, Manners and *Education.*

By JAMES NELSON, Apothecary.

Just as the Twig is bent the Tree's inclin'd. POPE.

LONDON:

Printed for R. and J. DODSLEY in Pall-mall;

And Sold by M. COOPER in Pater-noster-row. 1753.

Title page of James Nelson's book.

. . . A Child should not suck less than six Months, nor more than twelve; but that the Medium, that is, nine Months, is for the most part the best.

Hunger, Fatigue and Fretting, are the three most obvious things that impair a Mothers Health in nursing.

Now besides the latent causes of these Diseases, they have a very obvious one, *viz.* improper Food. For my own part I am convinc'd, even to Demonstration, that many Infants owe their Death to the Mistakes committed in this Point; and often to the false Practice of giving them Bread and Water only, and omitting that most salutary part the Milk. Milk (again I repeat it) is the Food of Nature; with that alone, to an Infant, we may do almost anything; without it, nothing.

The first Error usually run into, is the immoderate use of Spice and Sugar . . .

What is more common than to give young Children Lumps of Sugar to eat; yet what is more erroneous?

The Error next in rank to these, or rather a part of the same, as Sugar is greatly concerned in it, is, the Custom Parents have of giving Children Tea.

But farther; I am clearly of the Opinion, that the first change in Childrens Diet should be from Milk to Broth, and not from Milk to Meat. . . .

But besides this dreadful Habit (drinking) in the Mother or Nurse, there is a practice among the Vulgar still more shocking; and which must make every reasonable Creature shrink with Horror; that of giving Drams to the Children themselves, even while Infants.

To give Wine to Infants is a gross Error; and even to those who have pass'd that Stage, the Practice is very wrong.

Infants, at least for the first Year, have no Business with Malt Liquor at all; they ought not to take it; Milk or Water or both together, is their proper Drink; and if after this Age, these were made the Liquor to drink with their Food, it would be no worse for them. However not to be too rigorous in this respect, let Children after the first year, wash down their Victuals with light clear Small-Beer; and nothing beyond that for the first seven Years. In the second and third Septenary, the same Rule which has been laid down concerning Wine, should be observed in all strong Malt Liquors; they should be very sparingly used. Nothing is more dangerous than the Indulgence of Parents in this Point; for besides the many ill Effects already mentioned, 'tis coarse and vulgar; it clouds the Understanding, and renders young People unfit for Study. . . .

Let them too be wash'd every Day with cold Water, the Head and Limbs at least if not all over; and be carried out as much as can be into the open air.

They are not aware that to be fat is one thing, and to be healthy another, for bad Fat may be compared to ill-gotten Wealth, they both prey upon the vitals.

There is a Practice, particularly among the Great, shocking to Nature and to Reflection; that of using Paint. Paint is to the Face, what Affectation is to the Mind; as the one is a ridiculous Mimic of amiable Qualities we are Strangers to, so the other is a ridiculous Affectation of Beauty we cannot reach.

Notwithstanding a Child is advancing, I still recommend, that Milk and Water with Bread, or Milk-porridge, or Rice-milk, be its constant Breakfast.

Next to the proper kind of Food, nothing is more conducive to Health than a Habit of eating Meals regularly. Children who eat all day long are seldom healthy. . . .

Eating and Drinking are made too much the Business and the Pleasure of Life, to be consistent with either our Health or our Reason.

The last Caution I shall give to Parents relative to Health, is, the Danger of Nostrums and Quack-Medicines.

Severe and Frequent Whipping is I think a very bad Practice; it inflames the Skin, it puts the Blood into a Ferment, and there is besides, a Meanness, a Degree of Ignominy attending it, which makes it very unbecoming: still there may be Occasions which will render it necessary; but I earnestly advise that all the milder Methods be first try'd.

Pediatrics in Art

LIVING ROOM INTERIOR

JOHN RUHRÄH, M.D.
BALTIMORE

The artist Pinac, in the painting "The Virgin with the Child," depicted a beautiful interior. The picture is now in Turin. The child is small, rather of a microcephalic type, with a decidedly mature expression on its face. The picture gives one a good idea of the care of infants in the houses of the better class. Heat and ample ventilation are afforded by the open fire, and the whole scene indicates cleanliness and well being.

The Virgin with the Child.

News and Comment

GENERAL

Course in Pediatrics, New York Post-Graduate Medical School.—Under the direction of Prof. Roger H. Dennett, the New York Post-Graduate Medical School of Columbia University will give a condensed course in pediatrics for the physician. The course will be given one day weekly (Wednesday) for nine weeks (from Oct. 4 to Nov. 29, 1933), totaling sixty-three hours, at a fee of $25. For application forms, address the director, Prof. Roger H. Dennett, 303 E. Twentieth Street, New York City. The course is well developed and offers a varied program for the teaching of pediatrics.

Bequests and Donations.—The James Whitcomb Riley Hospital for Children of Indianapolis has been left the residue of the estate of the late Edmund Zoller, after a number of other bequests have been made.

The Children's Memorial Hospital of Chicago has been granted $10,000 from the estate of Mrs. Clara F. Bass.

DEATHS

We regret to record the death of Dr. A. Bratusch-Marrain of Vienna. Dr. Bratusch-Marrain was associated with Dr. Franz Hamburger for some years at the University of Graz and went with Professor Hamburger to Vienna where he was associated with him in the pediatric department of the University of Vienna. Dr. Bratusch-Marrain has contributed widely to pediatric literature, and it is sad to see a man of his attainments succumb at the early age of 35.

SOCIETY NEWS

Association Internationale de Pédiatrie Préventive. —The association will hold its third reunion on Sept. 15 and 16, 1933, at Luxemburg. The topics of discussion, with the introducers of each, are as follows: "Prevention of Infantile Paralysis," by Professor Rohmer of Strasbourg, and "How Can We Prevent Acute Affections in Nurslings?" by Professor Rott of Berlin. Information concerning this meeting can be obtained from Dr. D. Oltramare, A.I.P.P., 15 rue Lévrier, Geneva, Switzerland.

UNIVERSITY NEWS

Dr. John Zahorsky has been appointed director of the department of pediatrics at St. Louis University.

Abstracts from Current Literature

Anatomy and Physiology

BODY LENGTH AND ORGAN WEIGHTS OF INFANTS AND CHILDREN. J. M. COPPOLETTA and S. B. WOLBACH, Am. J. Path. **9**:55 (Jan.) 1933.

Charts showing the relation of age to body length and the weights of the heart, lungs, spleen, liver, kidneys, brain and all organs, based on 1,198 autopsies performed on infants from birth to 12 months of age are given. The article must be consulted in the original.

MOORE, Cleveland.

ROENTGENOLOGIC MEASUREMENT OF THE SIZE OF THE SELLA TURCICA IN INFANCY. ERICH SCHULZE, Arch. f. Kinderh. **93**:173 (May 15) 1931.

In this study the author has attempted to establish definite objective standards for the size of the normal sella turcica during infancy. The size of the sella in cubic millimeters was determined from roentgenograms of fifty normal infants, with the following results: 16 c. mm., from 1 to 3 months of age; 20 c. mm., from 4 to 6 months; 22 c. mm., from 7 to 9 months, and 23 c. mm., from 10 to 12 months. Tables are also presented to show the relation between the size of the normal sella and the circumference of the head and length of the body at various periods during infancy.

LEMASTER, Evanston, Ill.

RELATIVE SIZE OF THE HEART. OSKAR KIRSCH, Jahrb. f. Kinderh. **137**:185 (Nov.) 1932.

This is a long controversial article, opposing the views of Bernuth (*Fortschr. a. d. Geb. d. Röntgenstrahlen* **42**:368, 1930; *Ergebn. f. inn. Med. u. Kinderh.* **39**:69, 1931) and supporting those of the author's monograph (Grundlagen der Orthodiagraphischen Herzgrössen und Thoraxbreiten-Beurteilung im Kindesalter, Berlin, 1929).

CLAUSEN, Rochester, N. Y.

A PREVIOUSLY UNKNOWN BLOOD ELEMENT. A. EDELMANN, Wien. klin. Wchnschr. **44**:795 (June 19) 1931.

The author succeeded in isolating from the blood of normal persons morphologically and biologically well characterized bodies. They represent the fourth element of the blood in addition to the erythrocytes, leukocytes and blood platelets. Because of their mobility the author wishes to apply the name "kinetocytes."

These bodies appear dark under oil immersion with a dim condenser, as contrasted with the blood platelets, which are light-refracting. Neutral red solution stains them brown in the center and does not deprive them of their motility. They number from 1 to 2 in the field of vision, about 30,000 in a cubic millimeter. This number is not influenced by food intake, while the number of hematocones discovered by H. F. Müller in 1896 increases considerably after a meal, especially after the ingestion of fat. O. Naegeli is of the opinion that hematocones consist of fat particles, while the bodies discovered by the author are genetically different. On the basis of his observations the author offers the hypothesis that they play an important rôle in the coagulation of blood.

ABT, Chicago.

HUMAN SKIN CAPILLARIES: THEIR DEVELOPMENT AND SIGNIFICANCE IN CHILDREN. W. H. STEFKO, Časop. lék. česk. **70**:1301 (Sept. 25) 1931.

The author reviews the literature that has been published since Müller showed the variations of capillaries in various diseases. A classification of the funda-

mental types of capillaries is given with sketches. A capillaroscopic study of 241 backward, medium and advanced school children is reported. The backward school children are more likely to have hypoplastic capillaries, and normal forms were four times as prevalent in the good scholars. Of 45 cases of endocrine disturbance (19 of infantilism totalis, 6 of eunuchoidism, 6 of dystrophia adiposogenitalis and 14 of hypothyroidism with myxedema) in only 6 were normal capillary structures found (in 2 cases of infantilism and in 4 of dystrophia adiposogenitalis). Some degree of capillary hypoplasia is often associated with delayed growth and differentiation, as appears in dystrophic infantilism. Incomplete ectodermal capillary formation seems to be associated with thyroid hypofunction.

STULIK, Chicago.

CAPILLAROSCOPY IN SCHOOLS. W. F. STEFKO, Časop. lék. česk. **70**:1337 (Oct. 2) 1931.

The author feels that more definite knowledge and classification of capillaroscopic findings is essential before definite diagnostic value can be attained in endocrine disturbances and in tuberculosis, in which aplastic forms are often found. The findings of Jaensch in his study of several thousand school children are cited. His figures correspond generally with those of the author, and show 60 per cent aplastic forms in the backward children, 30 per cent in mediocre pupils and 21 per cent in good students. The highest percentages were found in the "Hilfsschule" for backward children.

STULIK, Chicago.

Pathology, Bacteriology and Biochemistry

SPOROTRICHOSIS: ITS TRANSMISSION TO PLANTS AND ANIMALS. RHODA W. BENHAM and BEATRICE KESTEN, J. Infect. Dis. **50**:437 (May-June) 1932.

This is an excellent article on the pathogenicity of certain fungi in man, animals and plants. Sporotrichum schenckii was the species studied. It produced in the monkey a clinical picture of nodular sporotrichosis such as that seen in man. The disease can be transmitted from human beings to carnations. Though the organism lives as a saprophyte in this flower, it retains its virulence for animals.

TOOMEY, Cleveland.

SOLUBLE SPECIFIC SUBSTANCES FROM YEASTLIKE FUNGI. H. D. KESTEN and E. MOTT, J. Infect. Dis. **50**:459 (May-June) 1932.

Kesten points out that a soluble specific polysaccharide fraction can be prepared from yeastlike fungi. He was successful in ten instances. By precipitin, absorption and other serologic tests, he proved the specificity of other fungi. He believes that the evidence indicates that Monilia albicans, M. psilosis and Endomyces albicans are strains of one species of M. albicans.

TOOMEY, Cleveland.

NONSPECIFIC ORCHITIS AND KERATITIS IN EXPERIMENTAL SYPHILIS. E. E. ECKER and L. A. WEED, J. Infect. Dis. **50**:484 (May-June) 1932.

These authors report that a spontaneous orchitis occurs when testicular tissue is transferred from rabbit to rabbit which is caused by Bacillus proteus (H type). They believe that this fact calls for some care in the interpretation of results in syphilis in rabbits.

TOOMEY, Cleveland.

INHERITANCE OF RESISTANCE AND SUSCEPTIBILITY TO INFECTIOUS ABORTION. MIGUEL MANRESA, J. Infect. Dis. **51**:30 (July-Aug.) 1932.

This is an interesting article from the standpoint of transmission of mendelian factors. The author concludes that there is an inheritable predisposition for

susceptibility or resistance to infectious abortions, and that this susceptibility or resistance characterizes each rabbit strain. These factors are probably transmitted through "a single pair of mendelian factors." Active immunization confers transitory immunity.

TOOMEY, Cleveland.

THE IMPORTANCE OF TEMPERATURE ON THE SURVIVAL TIME OF BACTERIA IN ACID FOODS. PAUL J. BEARD and J. P. CLEARY, J. Prev. Med. **6**:141 (March) 1932.

"It is well established that rise in temperature results in increased bactericidal efficiency of the hydrogen ion. It is conceivable that the hydrogen-ion concentrations rapidly bactericidal at ordinary temperatures might allow the survival of organisms over appreciable periods under conditions of lowered temperatures.

"Such a possibility has a practical bearing in relation to infection by means of foods whose sole method of preservation and treatment lies in low temperature storage. Citrus fruit beverages furnish a specific example, raw untreated orange juice being, for instance, served as a routine health measure in many public schools, sanitariums and other institutions."

Several strains of Eberthella typhosa, of Shigella dysenteriae and of staphylococci were selected as test organisms. Market orange juice, sterilized by filtration through Chamberland L3 candles, was seeded with these organisms and kept at various temperatures.

Organisms of the intestinal group may survive for seven days when kept at a temperature of — 4 C. and at p_H 3.5.

This indicates an appreciable inhibition of the bactericidal effect of the hydrogen ion by low temperatures. It follows that the preparation and handling of such foods should be subject to as rigid regulations and inspection as are applied to other types of food.

TURNER, Chicago.

THE SERUM TREATMENT OF EXPERIMENTAL STREPTOCOCCAL INFECTION. W. I. PENFOLD and HILDRED M. BUTLER, M. J. Australia **1**:717 (May 21) 1932.

The authors find that the following results are obtained in mice treated with commercial antistreptococcic serums, either as a prophylactic measure or as a method of treatment after the inoculation with streptococci: First, they may be unaffected by the use of the serum. Secondly, in a few cases they may be benefited by the serums. Thirdly, they may be definitely injured by it.

GONCE, Madison, Wis.

TWO GROUPS OF ATYPICAL DYSENTERY BACILLI. MARGOT MCKIE and F. M. BURNET, M. J. Australia **1**:760 (May 28) 1932.

Two groups of dysentery bacilli from patients with infantile dysentery studied in Melbourne hospitals are described as races U and T of Bacillus dysenteriae (Flexner). They are distinguished by their serologic and bacteriophage reactions from other Flexner strains.

GONCE, Madison, Wis.

GASEOUS EXCHANGE AND WATER ELIMINATION OF A RABBIT GIVEN INJECTIONS OF TETANUS TOXIN. ANDRÉ MAYER and G. NICHITA, Ann. de physiol. **7**: 333, 1931.

A rabbit was given injections of tetanus toxin. From the first spasm to the time of death it lost 164 Gm. in weight. The temperature increased until just before death. The respiratory quotient averaged 0.75. The water-dioxide quotient was 1.4. The respiratory gaseous exchange increased before the convulsions occurred. There appeared to be heat produced directly by the effect of the tetanus toxin itself besides the heat caused by muscular contractions. The elimination

of water did not appear to have any effect in regulating the temperature. It seemed to increase progressively and parallel to gaseous exchange.

BUDSON, Ann Arbor, Mich.

CHEMICAL PROCESSES IN THE COURSE OF CELL DIVISION. LOUIS RAPKINE, Ann. de physiol. **7**:382, 1931.

The author found that cell division was related to the quantity of chemical substances containing the — S H group. The amount of these chemical bodies varied during the course of cell division in the egg of the sea urchin and in various protozoa studied. The substance containing the — S. H group increased before cell division; it was due apparently to the breakdown of protein. The appearance of lactic acid was linked with the increase of the sulphydrated substance. It was possible to arrest cell division by the addition of heavy metals and to reestablish this division by the addition of substances containing the — S H radical.

BUDSON, Ann Arbor, Mich.

EXPERIMENTAL INVESTIGATIONS ON THE INFLUENCE OF THE FEMALE SEX HORMONE ON BLOOD REGENERATION. P. F. NIGST, Schweiz. med. Wchnschr. **62**:1156 (Dec. 10) 1932.

Using the placental extract, "Progynon," Nigst was able to accelerate regeneration of hemoglobin in female dogs. The phenomenon was observed in both normal and castrated animals.

WOLMAN, Philadelphia.

BLOOD PRESSURE-REDUCING EFFECT OF TONSIL EXTRACT. C. BISCHOFF and K. WEINAND, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:647 (July 22) 1932.

Bischoff and Weinand report the chemical extraction of acetylcholine from human tonsils. The extract was found to have a strong reducing effect on the blood pressure of cats. Since Bischoff, Grab and Kapfhammer have shown that acetylcholine is contained in many other organs, the authors think that the mere presence of this blood pressure-reducing substance in the tonsils does not justify designating the latter as glands of internal secretion.

EDITOR'S ABSTRACT [ARCH. OTOLARYNG.]

POSSIBILITY OF INFECTION WITH THE BACILLUS OF BANG THROUGH THE SKIN ROUTE: THE HISTOPATHOLOGIC PICTURE IN THE GUINEA-PIG. J. STOLZ and M. STOLZ-SUTORIS, Časop. lék. česk. **70**: 849 (June) 1931.

The authors were able to produce typical agglutination reactions by the inoculation of guinea-pigs in the following manner: (1) feeding of emulsion of Bang's bacillus, (2) subcutaneous injection of the emulsion, (3) application of the emulsion to skin which was shaved and traumatized (just shaved skin after a depilatory was used, skin scrubbed to remove protective fat and after prolonged ischemia) and (4) application to the skin with no previous preparation. The histopathologic picture is that of a chronic hyperplastic infection localized to the reticulo-endothelial structures, that is, the spleen, the lymph glands, the bone marrow and the liver. This group of diseases includes Malta fever, leishmaniasis, malaria and recurrent fever.

STULIK, Chicago.

Nutrition

MECHANISM OF CALCIFICATION IN THE HEART AND AORTA IN HYPERVITAMINOSIS D. ARTHUR W. HAM, Arch. Path. **14**:613 (Nov.) 1932.

Groups of rats were fed large doses of activated ergosterol for varying periods of time. Individual rats were killed at different intervals before and after calcification of the soft tissues occurred, and microscopic sections of the aorta

and other organs were studied. Large, single doses produced massive calcification of the heart and aorta within forty-eight hours. Since sections of the tissues taken twenty-four hours after beginning administration showed no abnormal changes, the calcification does not appear to depend on degenerative changes in the tissues. The rapidity of formation and the massiveness of the calcification suggest that the prime factor is the inability of the serum to retain all its calcium in solution. This precipitation of calcium might depend on such factors as the tension of carbon dioxide, the addition of other ions or an altered ratio between diffusible and nondiffusible calcium. LEMASTER, Evanston, Ill.

COMPARISON OF APRICOTS AND THEIR CAROTENE EQUIVALENT AS SOURCES OF VITAMIN A. AGNES FAY MORGAN and EVELYN O. MADSEN, J. Nutrition **6**:83 (Jan.) 1933.

According to biologic tests, the carotene of apricots is almost completely convertible to vitamin A by rats. The loss of vitamin A in the dried fruit was found to be from 50 to 75 per cent. MUNNS, Winnetka, Ill.

COPPER, IRON AND MANGANESE CONTENT OF FISH. THOMAS B. PARKS and EMBREE R. ROSE, J. Nutrition **6**:95 (Jan.) 1933.

Twenty species of fresh fish, some caught in Lake Champlain and others bought in the market, were studied. Part were salt-water fish, and part were fresh-water fish. The copper content of both fresh-water and salt-water fish averaged about 2.5 mg. per kilogram of moist material. Salt-water fish contained about 12 per cent more iron than fresh-water fish, and fish having dark-colored tissue had 75 per cent more iron than those with light-colored tissue. Manganese in small amounts was found in the muscle tissue of all species examined.

MUNNS, Winnetka, Ill.

VITAMIN A CONTENT OF YELLOW-TISSUED AND WHITE-TISSUED APPLES. M. T. POTTER, J. Nutrition **6**:99 (Jan.) 1933.

Two varieties of yellow-tissued and one of white-tissued apples were studied. All were found to contain almost an equal quantity of vitamin A according to biologic tests, and all had a potency of vitamin A equal to that of orange juice.

MUNNS, Winnetka, Ill.

SUBSTITUTE BREAST FEEDING. CHARLES GILMORE KERLEY, M. J. & Rec. **136**:371 (Nov. 2) 1932.

The greatest achievement in the feeding of infants denied breast milk has been the establishment of high grade cow's milk, evaporated milk and powdered milk, safe for infant nutrition. The author divides the infants into two groups: 1. Infants who have normal gastro-intestinal systems, with average digestive capacity. These infants were fed on fresh milk, sugar, starch and water, from 60 to 80 calories per pound (0.5 Kg.) of body weight being allowed. An interval of four hours was left between the feedings. They were given orange juice and tomato juice in every case. Green vegetables were not allowed in the early months. 2. Infants presenting problem cases, including infants who are difficult to feed from birth, infants fed unsuitable food, infants irregularly fed, infants suffering from some malformation and sick infants. Suggestions given for these patients were: the use of evaporated or dried milk; lavage of the stomach in cases of anorexia, vomiting and mucous gastritis; the administration of atropine in cases of vomiting and to all infants subject to frequent attacks of colic. Cod liver oil and orange juice must be added cautiously. WILLIAMSON, New Orleans.

A Few Generalizations on Modern Infant Feeding. Marshall Carlton Pease, M. J. & Rec. **136**:372 (Nov. 2) 1932.

A large group of the population depends on the milk supply not only for life but for health. The infant requires a suitable food given in sufficient quantities at regular intervals. Cow's milk is still, on the whole, the most suitable food after breast milk. If it is clean and properly handled, cow's milk is the food of choice. Infants have more difficulty in digesting carbohydrates and fats, and each patient varies considerably in his capacity for digesting both fats and sugars. It is important in the earlier stages not to overfeed the baby. The author advocates that the feeding of cereals be begun between the third and fifth month, and that a little later the green vegetables be given. Any infection in a small child will retard gain in weight. The premature infant, the allergic child or the child suffering from a chronic digestive disturbance is not discussed in this article. Infant feeding is not a complicated problem, but the main essentials must be understood and carried out with care. A clean milk, a good sugar, an understanding mother and a healthy baby are the principal elements for success in infant feeding.

Williamson, New Orleans.

Biophysiological Appetizers in the Nutrition of the Child. George Dow Scott, M. J. & Rec. **136**:376 (Nov. 2) 1932.

Hunger, thirst and fatigue have long been recognized by pediatricians as biophysiologic agents in the growth, development and health of children; as they stimulate the desire for food, fluids and rest, they may be styled appetizers. In the normal child, strong hunger contractions are undoubtedly biologic evidence that the stomach is in proper condition to receive food. These contractions recur at intervals as long as the stomach is empty. In the sick child, these intervals are prolonged, and hence the feeding period should be extended beyond the maximum limit. The physiology of these hunger contractions and their influence on other parts of the body are then discussed. The gastric secretion obtained through stimuli of sight, taste and smell is usually referred to as psychic, or better as appetite, juice. Muscular activity and bathing may increase the appetite in some children. The author then points out the important part that environment and the emotions play in the health and happiness of the child, and also the difference in the quantity and in the quality of the gastric juice under certain conditions. The hunger urge in children is shown symptomatically by a more or less uncomfortable feeling referred to the region of the epigastrium, the intensity and persistence of which depend on the degree of body activity and the rate of metabolism.

Williamson, New Orleans.

National Consumption of Carbohydrates in Relation to Disease. J. H. P. Paton, Edinburgh M. J. **39**:556 (Sept.) 1932.

In all countries the consumption of sugar is rapidly increasing. Paton warns that sugar as it is now used is not a natural food, but that it has been refined by a series of chemical processes, such as treatment with sulphurous acid, neutralization with lime and repeated evaporation in vacuum pans, so that it is totally void of vitamins. The starch-bearing foods are denaturized by the miller so that fat-soluble vitamins are removed with the germ. The present cheapness of sugar has somewhat displaced fats from the dietary. The amount of sugar used is excessive. More than 100 pounds (45 Kg.) per person was consumed annually in a boarding school surveyed. In routine examination of urine from healthy school girls sucrose was found in 50 per cent of the specimens, and none was found when sugar was kept out of the diet. The inverting power of the gastro-intestinal juices is limited, and it is probably inadequate for the person who is ingesting the present average of 100 pounds a year. As there has been no reduction in the intake of starchy foods, Paton assumes that most of the sugar that is eaten is surplus. Half of the sucrose eaten is taken as levulose into the portal blood. Fruits contain some levulose but usually also dextrose, and there are other contents, such as salts,

acids and vitamins which help to dilute the levulose. Evidence seems to point to the fact that levulose is not used by the human economy; it is therefore useless, and may even have some toxic action. It strains the hepatic activity, which does not occur when natural sugars high in levulose, such as fruits and honey, are ingested.

Paton believes that an excess of carbohydrates has increased the incidence of severe catarrhal diseases, acidosis in children, rickets, dental caries and diabetes.

NEFF, Kansas City, Mo.

RELATIONS BETWEEN THE WEIGHT OF BREAST-FED INFANTS, THEIR ORDER OF BIRTH, AND THE YIELD AND COMPOSITION OF THEIR MOTHER'S MILK. H. S. HALCRO WARDLAW, M. J. Australia **2**:564 (Nov. 5) 1932.

From a study of 275 babies the author concludes that: The earlier infants of a family of moderate size tend to weigh less than those born later. For breast-fed infants a similar relation between weight and order of birth holds up to the age of about 6 months. The growth of the infants of primiparas tends to be retarded more during the first four weeks of life than the growth of later children. The relatively heavier, later children of a family have available a more plentiful supply of breast milk than the earlier, lighter children. There is no appreciable correlation between the concentration, as distinct from the quantity, of the supply of breast milk and the relative weights of breast-fed infants.

TENNEY, Madison, Wis.

PHOTOCHEMISTRY OF VITAMINS A, B, C AND D. F. P. BOWDEN and C. P. SNOW, Nature, London **129**:720, 1932.

The monochromatic irradiation of B-carotene, vitamin B_1, hexuronic acid, narcotine, ergosterol and calciferol is described. The absorption spectrum of the substance was studied, and then it was irradiated with an intense beam of light of a wavelength similar to that of an absorption band selected after consideration of the spectroscopic data. When applied to a suitable provitamin, monochromatic irradiation with light of the correct wavelength produces maximum yields of the vitamin. When the destruction by irradiation of the biologic activity of a vitamin is effected by the elimination of an absorption band, the activity can be linked up conclusively with the band. Thus, irradiation of a solution of B-carotene in cyclohexane produced the band at 3,280 angstroms characteristic of vitamin A. Irradiation of a Jansen and Donath Vitamin B_1 preparation by the mercury line 2,537 angstroms reduced the intensity of the band at 2,600 angstroms and destroyed the activity of the sample, as tested by observation of the heart beats of rats. Similar but less conclusive studies involving vitamins C and D are reported.

KUGELMASS, New York.

HALIBUT LIVER OIL AS A SOURCE OF VITAMIN A. J. A. LOVERN, Nature, London **129**:726, 1932.

Lovern has confirmed previous reports that the liver oil of Hippoglossus vulgaris is from 50 to 100 times as rich in vitamin A as cod liver oil. However, wide irregularity in potencies was noted, variations observed ranging from 30 to 1,600 blue units. Extensive experiments in search of the causes of these fluctuations are in progress.

KUGELMASS, New York.

THE AMOUNTS OF KETONE BODIES IN THE URINE OF ARTIFICIALLY FED BABIES, BOTH HEALTHY ONES AND THOSE WITH A NUTRITIONAL DISTURBANCE. M. GIUFFRE and G. MANNA, Riv. di clin. pediat. **30**:1389 (Nov.) 1912.

Quantitative estimations were made of the amounts of acetone diacetic acid and *beta*-oxybutric acids in the specimens of urine of eleven infants with intestinal intoxication and six healthy and four undernourished infants, all artificially fed. Many of the infants excreted no acetone bodies.

The excretion by healthy infants averaged 3.2 mg. of acetone, 0.4 mg. of diacetic acid and 0.4 mg. of *beta*-oxybutyric acid per hundred cubic centimeters of urine. For undernourished infants, the excretion tended to be greater, averaging 4.2 mg. of acetone, 0.9 mg. of diacetic acid, and 0.4 mg. of *beta*-oxybutric acid. The excretion in the infants with intestinal intoxication showed considerable variation. Some infants excreted no acetone bodies. The maximum excretion per day was 15.7 mg. of acetone, 4.1 mg. of diacetic acid and 3.2 mg. of *beta*-oxybutyric acid.

A 4 year old child with vomiting excreted 525 cc. of urine in one day, which contained 54.9 mg. of acetone, 4.6 mg. of diacetic acid and 37.6 mg. of *beta*-oxybutyric acid.

HIGGINS, Boston.

PUERPERAL GALACTOPHORITIS AND ITS EFFECTS ON NURSING. S. MAGNI, Riv. di clin. pediat. **30**:1419 (Nov.) 1932.

Galactophoritis is rather frequent in nursing mothers. It should be diagnosed and treated early. If it is neglected, it may result in abscesses of the breast or severe septicemia and possibly predispose the mother to cancer. Localized or generalized pyogenic infections and, possibly, digestive disturbances and malnutrition may develop in the child.

Treatment consists mainly in freeing the milk ducts from the pathogenic products by thorough emptying of the breasts.

Prophylaxis consists in proper hygiene and cleanliness and in the avoidance of pressure on the breasts, especially about the nipples, from elastic or stiff fabrics such as are used in some brassieres.

HIGGINS, Boston.

SOME CHEMICAL AND PHYSICOCHEMICAL CHANGES OF THE MILK OF WOMEN PUT ON A DIET LOW IN CHLORIDES. R. PACHIOLI and R. GRANATIERO, Riv. di clin. pediat. **30**:1457 (Nov.) 1932.

Observations were made on the percentage of chlorides, the percentage of lactose and the freezing point of the milk of eleven women before and after they had been put on a low salt diet. The average figures are given in the following tabulation:

	△T	Sodium Chloride, per Cent	Lactose, per Cent
Normal diet	0.568	0.91	6.41
Low salt diet	0.564	0.53	6.82

The lower salt content of the milk is compensated for by a higher lactose content, so that there is little or no change in the osmotic equilibrium of the milk.

HIGGINS, Boston.

DEFICIENCY IN VITAMIN A. ANTONIO CARRAU, Arch. de pediat. d. Uruguay **3**:249 (June) 1932.

A deficiency in vitamin A produced by a regimen poor in fat substances is not an uncommon experience in the present-day infant. The author believes that many of the cases of dystrophy so frequently encountered in the hospital should be carefully investigated from the standpoint of a deficiency in vitamin A.

The author reports three cases in which the patients ranged in age from 5½ to 10 months. All of the patients showed a pathologic condition of the eyes. The feeding of a high fat diet adequate in vitamin A caused the condition of the eyes to clear up rapidly.

SCHLUTZ, Chicago.

Prenatal Conditions

CONGENITAL HEART DISEASE (CLINICAL TYPES AND DIAGNOSIS). HOWARD B. SPRAGUE, New England J. Med. **207**:525 (Sept. 22) 1932.

This condition represents about 5 per cent of the cases of heart disease of childhood. During the first year of life, the diagnosis is far more difficult, as the characteristic murmurs frequently do not appear during the first year.

Dr. Maude Abbott reports eight hundred and fifty cases studied pathologically, and divides the lesions into a total of sixty-four subgroups. The principal groups are the following:

1. Ectopia cordis. The heart may be in the abdominal cavity or outside the wall of the chest. The maximum duration of life has been noted as fifteen months and the mean duration as one month.

2. Congenital idiopathic hypertrophy. The diagnosis of this condition is made by exclusion. The maximum reported duration of life is four years and the mean, sixteen months. The diagnosis is made by finding a generally enlarged heart in a young child in whom there is no evidence of pericardial effusion or valvular disease.

3. Dextrocardia. The condition can be proved by means of the electrocardiogram which shows a complete inversion of the waves in lead I and a transposition of leads II and III. The mean duration of life in Abbott's series is thirteen years and the maximum, forty-six.

4. Interventricular septal defect (Roger's disease). The usual defect in the septum between the ventricles is situated high up in the fibrous portion of the septum. The diagnostic signs consist of a rough systolic murmur and thrill in the midsternal region or at the third and fourth intercostal spaces near the sternum. The electrocardiogram frequently shows some degree of right axis deviation. The oldest patient reported in Abbott's series and studied at necropsy was 44 years of age, and the average was 13 years and 9 months.

5. Patent ductus arteriosus. Milder degrees of patency of the ductus arteriosus are common. The condition occurred in ninety-seven cases in Abbott's series. The common finding is an arteriovenous shunt. Therefore, the patients are not cyanotic. The diagnosis is made by finding a continuous murmur and thrill, maximal at the second left interspace near the sternum. The mean duration of life is thirty-three and one-fourth years.

6. Subaortic and aortic stenosis. It is unusual to find rheumatic aortic stenosis in young children, and the discovery of the characteristic systolic murmur and thrill at the second right interspace in an infant or young child suggests strongly the diagnosis of congenital stenosis of the conus of the left ventricle or the aortic valve.

7. Tetralogy of Fallot. The tetralogy consists of stenosis of the infundibulum of the right ventricle and of the pulmonary valve, a defect in the interventricular septum, dextroposition of the aorta and enlargement of the right ventricle. The average duration of life is eleven and one-half years. There is marked enlargement of the right ventricle, as shown by physical examination, roentgenograms and electrocardiograms, and cyanosis with clubbing of the fingers and toes. The heart may show a rough systolic murmur and thrill, loudest at the second left interspace near the sternum.

8. Coarctation of the aorta (adult type). During the development of the ductus arteriosus there may be a narrowing in the descending aorta at the point where the ductus leaves it. This narrowing may be of sufficient degree to cause marked circulatory changes. The heart tends to hypertrophy when forced to contract against this obstruction. There is a hypertension of the upper part of the body, with a low blood pressure in the arterial system below the coarctation. This produces a paradoxical condition of the blood pressure in the arms and legs.

GENGENBACH, Denver.

TWO ESOPHAGEAL CASES. J. V. BOHRER, New York State J. Med. **32**:901 (Aug. 1) 1932.

A newly born infant vomited immediately after each feeding. The vomitus was alkaline and contained no curds. A roentgenogram showed that the esophagus ended in a blind pouch at the level of the bifurcation of the trachea. The lower portion of the esophagus connected the trachea with the stomach so that liquid given through a gastrostomy opening caused severe coughing and cyanosis. The child lived for two weeks. At autopsy no congenital anomalies other than the one mentioned were found.

AIKMAN, Rochester, N. Y.

ANATOMIC STUDY OF "KLIPPEL-FEIL" SYNDROME. A. FEIL, A. LEBLEU and H. FISCHER, Rev. d'orthop. **19**:531 (Sept.-Oct.) 1932.

The Klippel-Feil syndrome is characterized by:

1. The cervical vertebrae are fused into a mass along with the upper dorsal vertebrae.

2. An upper cervical spina bifida is a constant finding.

3. The thoracic cage is elevated; a "cervical thorax" is present.

Clinically, there is a short neck; the head is planted directly on the thorax; the appearance of the patient is that of a "man without a neck." The hair at the base of the scalp lies in a horizontal line; movements of the head are limited, especially in the lateral motions. Movements are mechanical, but not painful. The arms are in disproportion with the trunk; they are longer than normal. The back is kyphotic or scoliotic.

Three complete autopsies have been reported in the literature previously. The case reported is the fourth in which a complete postmortem examination was made.

A child, 15 months of age, born at term, after a normal labor, was noticed at birth to have a short neck; there was a tumor 7 cm. in diameter, slightly tender, fluctuant, translucent and surmounted by two smaller tumors. The right scapula was higher than the left; abduction of the arms was difficult. There was a bilateral coryza; the eyes were apparently blind.

The child was born of healthy parents, who had previously had four healthy children. Since the tumor appeared to be increasing, operation was decided on. Two curved incisions were made, one on either side of the pedicle; the skin was retracted, and the pedicle ligated. The following day the temperature rose to 39 C. (102.2 F.), and the child died.

Roentgenographic examination was made of the neck both before dissection and afterward. The transparency was normal. The upper part of the cervical region appeared wider than usual; the cervical column was short, and the top of the thoracic cage appeared to press directly on the base of the head.

Anatomically, there was a large opening in the cervicodorsal region through which the tumor presented. The vertebrae were an atrophic mass; they were united with one another. The whole mass equaled four thoracic vertebrae in height. No atlas or axis was found. Two or three ribs apparently arose near the base of the mass. The mass appeared to be composed of all of the cervical and two or three of the thoracic vertebrae. The lateral foramina were poorly delineated, four being seen on each side, which were unequal and irregularly spaced. The ribs were normal. The right side of the thorax was higher than the left. The base of the skull rested on the mass. Below the mass the spine was normal. A roentgenogram of the dissected spine showed the remnants of six vertebrae.

Therefore, the etiology of the condition must be congenital malformation. The hypothesis that an infectious spondylitis of early infancy might be the cause can hardly be held.

RYAN, Vancouver, B. C.

REPORT OF TWO CASES OF PERSISTENT DUCTUS ARTERIOSUS. G. DAGNINI, Pediatria **13**:601 (Aug.) 1931.

The author reports two cases of persistent ductus arteriosus, that is, a failure of the common trunk of this duct to divide into aorta and pulmonary artery;

there was a continuation of its embryonic state, in which the common trunk served a double function of aorta and pulmonary artery.

Both cases occurred in girls; one died at birth and the other lived only two days. In one case, the anomaly was associated with absence of all the septums of the cardiac cavities; in the second case, no other anomaly was present.

The author calls attention to the rarity of this condition, pointing out that only fifty-three cases have been reported in the medical literature.

SIGNORELLI, New Orleans.

CONGENITAL ATRESIA OF THE BILE DUCTS AND "SITUS VISCERUM INVERSUS." P. SCHIAPARELLI, Riv. di clin. pediat. **30**:903 (July) 1932.

A report of a case with necropsy observations is given for a child, aged 5 months, with congenital atresia of the bile ducts and transposition of the viscera. If these two congenital abnormalities have the same cause, it must be traced back to a teratogenic defect occurring in the third or fourth week of embryonic life.

HIGGINS, Boston.

TREATMENT OF PREMATURE INFANTS AND CONGENITALLY WEAK INFANTS. TOMAS SALA SANCHEZ, Pediatria españ. **21**:494 (June) 1932.

The author believes that premature and congenitally weak infants should primarily be cared for in maternity hospitals, children's clinics and child welfare centers, where it is entirely possible to give the care they require.

It is not easy to arrive at exact conclusions regarding the frequency of premature birth, as there does not now exist unanimous agreement among the pediatricians and obstetricians or even among pediatricians themselves as to the signs of maturity in the newly born. The author is in agreement with most authorities that a birth weight under 2,500 Gm., regardless of the age of the fetus, classifies the newly born as premature.

In the treatment of the premature infant, three fundamental things must be stressed: regulation of heat, protection against infection and adequate nutrition. The author is opposed to the use of all forms of incubators. He considers them useless and even harmful. He recommends instead the following procedure: The infant is placed in a bath, ranging in temperature from 35 to 40 C. (95 to 104 F.) and kept there for about five minutes. It is then wrapped completely in cotton and placed in a half-open box and kept in a constant temperature of about 36 C. (106 F.). Heat is supplied to the box by means of hot water bottles. A competent nurse is placed in charge whose duty it is to watch the temperature and to change the hot water bottles when necessary.

Infection is guarded against by having the crib or box surrounded by moist gauze and by having the attending personnel carry out the most rigorous aseptic technic. Special gowns and masks soaked in glycerinated water are worn. The infants are not bathed or weighed oftener than every three or four days. Rigid hygiene of the skin is carried out.

Breast milk is the only aliment which should be considered for the premature infant. If this is not available, the author prefers the use of buttermilk mixtures or fat free milk mixtures, with the addition of malt sugars. Dried or evaporated breast milk is also effective. As soon as the infant can suck and swallow well, it is placed directly at the breast. The author recommends small and frequent feedings, as many as ten feedings in the twenty-four hours.

The author believes that there is no fixed rule which regulates the caloric intake of the premature infant. The aliment is in close relationship to the susceptibility of the infant and depends on the amount of reserve material with which the infant came into the world. In general, a caloric intake of between 140 and 160 calories per kilogram of weight is required for the first month and from 150 to 190 calories per kilogram for the second month.

The author reports a mortality of from 11 to 17 per cent after the first few days. Including these, his total mortality is about 49 per cent. The mortality is confined largely to infants weighing under 2,000 Gm. Meningeal hemorrhages caused the largest number of deaths. Bronchopneumonia ranked second as a cause.

SCHLUTZ, Chicago.

CAUSES OF CONGENITAL ATRESIA OF THE SMALL INTESTINE. FRITZ LOESSING, Arch. f. Kinderh. **97**:1 (July 22) 1932.

This is a report on a case of atresia at the junction of the jejunum and ileum, in a premature infant dying on the third day of life. The author briefly discusses the various theories concerning the causes of congenital obstruction of the small bowel which have been proposed by different authors. Serial sections of the bowel in the case reported are interpreted as indicating trauma to the bowel before the fifth fetal month as the etiologic agent producing the atresia.

LEMASTER, Evanston, Ill.

ILLUSTRATIVE CASE OF ARACHNODACTYLIA. A. KOLLMANN, Arch. f. Kinderh. **97**:206 (Oct. 4) 1932.

Only twenty-five cases of arachnodactylia have been reported in the German literature. The case reported in this article was in a girl, 2 years and 8 months of age. The characteristic findings consisted of increased length and slenderness of the extremities, especially the fingers and toes, underdevelopment of the musculature, marked laxity of the ligaments and deficiency in subcutaneous fat. All of the reported cases of this condition are reviewed. Other frequent findings include increased height, funnel breast, flat feet, a spurlike process on the calcaneum and anomalies of the eyes, ears and heart. Various etiologic theories are discussed.

LEMASTER, Evanston, Ill.

SPINA BIFIDA OF THE ATLAS. P. GEIPEL, Fortschr. a. d. Geb. d. Röntgenstrahlen **42**:583 (Nov.) 1930.

Nine cases of spinal bifida of the atlas were found in a total of 260 macerated vertebral columns, an incidence of 3.5 per cent. The separation varied from a simple fissure to a cleft 6 mm. wide, and was localized mostly from 2 to 3 mm. to the left of the midline. The condition has not as yet been observed roentgenologically in the living.

[AM. J. ROENTGOL.]

DYSOSTOSIS CRANIOFACIALIS. M. I. SCHOR, Ztschr. f. Kinderh. **53**:103, 1932.

Dysostosis craniofacialis is characterized by malformations of the face and cranial vault. The cranial malformation is probably due to the premature ossification of the coronal or sagittal sutures. Clinical descriptions of the syndrome are given, and typical roentgenographic appearances of the skull are shown.

SHELLING, Baltimore.

AGENESIS OF THE EXTREMITIES. H. SYKORA, Časop. lék. česk. **70**:891 (June 19) 1931.

The author presents the report of a case of a girl, 2 months of age, with the following defects: The right femur is partially developed; the lower end simulates the lower end of the tibia. There are a great toe and three small toes. The right leg is replaced by a structure consisting of a short irregular mass, 5 cm. long, with a great toe and a rudimentary small toe. Both upper extremities are missing, although the shoulder girdle is well formed. The internal organs and the brain are essentially normal, though the cranium is relatively small. The author discusses the exogenous and endogenous factors in the development of agenesis, and considers the various theories promulgated.

STULIK, Chicago.

CASE OF AGENESIS OF THE SPLEEN. E. BEHR, Nederl. tijdschr. v. geneesk. **76:** 1490 (March 26) 1932.

The author reports a case of agenesis of the spleen in a girl 2½ years old. During a long period she had been observed in the hospital on account of congenital dextrocardia with stenosis of the pulmonary artery. This disturbance is very rare and—so far as the author could find—not mentioned in Dutch literature. Of all the cases in the literature (over thirty) a minute microscopic examination of the various organs has been performed in only a few. The author gives a detailed report of the present case. As in a great many of such cases, other disturbances in development could be found, especially ones of the heart and of the large vessels; perhaps a deficiency of the blood supply of the mesogastrium may be the cause of the agenesis of the spleen. Another cause for this disturbance has not yet been found.

VAN CREVELD, Amsterdam, Holland.

INTRA-UTERINE PARAPHIMOSIS. J. A. H. HERMANS, Nederl. tijdschr. v. geneesk. **76:**1494 (March 26) 1932.

A case is described in a new-born child in whom there was an erect penis, a swollen, deeply cyanotic glans, no prepuce and, instead of this, a bleeding, granulating rim. Recovery occurred without complications.

VAN CREVELD, Amsterdam, Holland.

CONGENITAL OCCLUSION OF THE ESOPHAGUS. P. KLUMPER, Nederl. tijdschr. v. geneesk. **76:**1666 (April 2) 1932.

The author observed two cases of this fairly rare condition, and gives the clinical symptoms and the results of the autopsy. In the first case, that of a girl, whose weight at birth was 4,020 Gm., there existed a total occlusion of the esophagus just above the bifurcation of the trachea. Immediately below this occlusion there was a narrow communication between the trachea and esophagus. The second child had a birth weight of 2,960 Gm.; in this case the lumen of the esophagus ended 2 cm. above the bifurcation of the trachea. From this place it continued as a narrow cord in the posterior wall of the trachea, becoming wider just below the bifurcation. In this case, there also was a communication between the trachea and the esophagus. The described anomalies of the esophagus are discussed, both from a phylogenetic and from an obstetric point of view.

VAN CREVELD, Amsterdam, Holland.

Diseases of the New-Born

TWO CASES OF RUPTURED LIVER IN THE NEWBORN. H. J. R. MCNITT, Am. J. Obst. & Gynec. **23:**431 (March) 1932.

Rupture of the liver in the new-born has seldom been described in the literature. The usual history of the case is that of an apparently normal child, who, within three or four days of birth, suddenly becomes ill and dies within a few hours. Actual rupture of the liver is the least common of intra-abdominal traumas. Death in actual rupture is postponed for three or four days, during which time a subcapsular hemorrhage is slowly taking form; a fatal intra-abdominal hemorrhage results. The usual cause of this condition is pressure or torsion of the fetal abdomen. Usually there is a history of definite external trauma. The two cases are presented to show the possible sequelae of antepartum manipulations.

ZIEGLER, Minneapolis.

BLOOD CYSTS ON THE HEART VALVES OF NEW-BORN INFANTS. SAMUEL A. LEVINSON, Arch. Path. **14:**810 (Dec.) 1932.

Small blood-filled cysts ranging in size from that of a pinpoint to that of a pinhead were found on the valve leaflets of the heart in 75 per cent of newly

born infants. The nodules occur with greatest frequency between the seventh month of intra-uterine life and the fifth month of extra-uterine life, and they become a rarity after two years of age. The authors favor the view that these nodules are formed by blood pressed into the endothelial clefts of the leaflets during systole, combined with adhesions of the endothelium at the opening of the cleft, causing a small cyst lined with endothelium and filled with blood which is later absorbed.

LeMaster, Evanston, Ill.

Chvostek's Facialis Sign in New-Born Infants. A. G. Mitchell and Frank E. Stevenson, J. A. M. A. **99**:1502 (Oct. 29) 1932.

In recent reports of infantile tetany or spasmophilia in new-born infants, the presence of Chvostek's facial phenomenon has been given much diagnostic importance. In fifty-five apparently healthy new-born infants, the facial sign was obtained consistently. Fifteen were examined for electrical reactions, and all were normal. The blood calcium in all but five was between 10 and 12 mg. In one it was 7.3 mg. and in four between 8 and 10 mg. Infantile tetany should not be diagnosed on the presence of Chvostek's facial phenomenon in the absence of characteristic electrical reactions and a lowered blood calcium.

Bonar, Salt Lake City.

The Successful Treatment of Respiratory Failure of the New-Born in the Drinker Respirator. Harry S. Snyderman, M. J. & Rec. **136**:379 (Nov. 2) 1932.

The treatment of asphyxia has a twofold object: (1) It should initiate respiration, and (2) it should also satisfy respiratory needs until normal breathing is established. The respirator and the principle on which it works are described. Nine severely asphyxiated infants were treated in the respirator; seven were completely relieved, and two died in spite of treatment, but the latter were found by spinal tap to be suffering from cerebral hemorrhage.

Williamson, New Orleans.

Feet of New-Born Infants. H. A. Sweetapple, M. J. Australia **1**:892 (June 25) 1932.

Examination of the feet of one thousand new-born infants shows that: Forty and four-tenths per cent of the feet examined occupied, at rest, positions which would be deformities if they persisted; the relative frequency of the various positions at rest bears no relation to the relative frequency of the various forms of congenital talipes; feet which possess a fixed deformity at birth can, without treatment, become normal after birth; the infant's foot is capable of much greater range of movement than that of the adult, and feet possessing a deficient range of movement at birth can acquire the normal range after birth.

Gonce, Madison, Wis.

Cerebral Hemorrhage in an Infant Before the Beginning of Labor. H. Nerson, Bull. Soc. d'obst. et de gynéc. **21**:519 (July) 1932.

The mother of the infant was a septipara, admitted to the hospital because of a diagnosis of placenta praevia. She had three uterine hemorrhages in two weeks, the last of which occurred the day of admission. Previous to this her pregnancy had been uneventful, and she had had no labor pains. The woman appeared very anemic; the cervix was dilated two fingerbreadths and was filled with placental tissue; the Wassermann reaction was negative. The fetal heart sounds were normal. Cesarean section was done under ether anesthesia. The infant was found in the uterus in the position of a breech presentation with the breech in the left iliac fossa posteriorly. The mother made an uneventful recovery and was discharged after four weeks.

The infant appeared to be a full term, physically normal child, and weighed 3,070 Gm. The heart tones were good at first, but the child failed to breathe, the heart rapidly ceased to beat, and death occurred. Necropsy showed petechial hemorrhages in the epicardium, pulmonary atelectasis, intense cerebral congestion and a hemorrhage into the posterior horn of the left lateral ventricle. The liver and spleen were of normal weight, and there was no evidence of syphilis. The family history of the mother was unimportant as regards similar conditions. The author regarded this case as evidence that intracranial hemorrhage may occur in an infant before the beginning of labor and therefore have no relationship to obstetric trauma.

GLASER, Rochester, N. Y.

A CASE OF RITTER'S DISEASE. H. ELIAS, Bull. Soc. de pédiat. de Paris **30**:434 (May 14) 1932.

Dermatitis exfoliativa neonatorum (Ritter's disease) is apparently a rare disease in France and Rumania, although several large series of cases have been reported from Germany. The case of a boy, 3 weeks old, afflicted with this condition, who died presumably of pyemia is reported. The disease is regarded by some writers as a form of pemphigus neonatorum and by others as a distinct entity.

BENJAMIN, Montreal, Canada.

ICTERUS OF THE NEW-BORN. A. GOLDBLOOM and R. GOTTLIEB, Rev. franç. de pédiat. **8**:177, 1932.

Simple icterus of the new-born is a physiologic state of hemolytic nature. The infant passes abruptly from a prenatal anoxemia to a postnatal state, which results in the hemolysis. In animals icterus resembling that in the new-born can be caused by a reproduction of the oxygen tension that simulates that found in the prenatal and postnatal state.

SANFORD, Chicago.

INTESTINAL OCCLUSION IN THE NEW-BORN. RAUL M. DEL CAMPO, Arch. uruguayos de med. **1**:237 (Nov.) 1932.

The author reports three cases. He believes the etiology to be a fetal peritonitis. Examination shows that the intestinal integument is atrophied, especially the muscular coat. The absolute symptoms are vomiting, no bowel movement, distention and peristalsis. Other occasional signs are elevation of temperature, oliguria and icterus. The prognosis is practically hopeless, although prompt surgical intervention might offer some chance in certain favorable cases.

SANFORD, Chicago.

PHYSIOLOGIC VARIATIONS OF RETICULOCYTES IN THE NEW-BORN: A STUDY OF TWO HUNDRED AND NINETEEN CASES. KATSUJI KATO, Folia haemat. **46**:377, 1932.

In the circulating blood of 199 normal newly born infants, irrespective of race or sex, the author found an average of from 1.4 to 1.9 per cent of vitally stainable reticulocytes, with the maximal limit of 5.5 per cent and the minimum of 0.1 per cent during the first thirty-six hours of life. When the percentage of reticulocytes was low during the first eighteen hours, there was a tendency toward an increase during the next eighteen to thirty-six hours, while high initial values were followed by a decline. The number of reticulocytes in some cases was observed to proceed parallel with the values for nucleated red cells. In 14 premature infants, the reticulocyte determinations showed about the same initial values and general decline to normal values in from four to seven days as in full term infants. The literature concerning supravital staining of erythrocytes is completely reviewed.

LEMASTER, Evanston, Ill.

Acute Infections

THE RATIONAL DOSAGE OF DIPHTHERIA ANTITOXIN. ERNEST GRUNDY, Brit. M. J. **2:** 1132 (Dec. 19) 1931.

The author reviews the history of the dosage of diphtheria antitoxin, expressing opinions on doses, from the minimum to the maximum. He discusses the advisability, as recommended by Parks, of a single adequate dose in the beginning with no repetition; he then reviews the results obtained following the use of various doses in certain hospitals in England and comes to the following conclusions:

1. The mortality in diphtheria varies almost inversely as the amount of antitoxin administered until an adequate dose is reached. 2. The average dose should be equal to that given in one of the hospitals, which varies from 14,500 units to 55,000 units for a primary intramuscular injection. The total varies from 40,500 to 145,000 units. 3. Intramuscular injection of antitoxin is a satisfactory method of administration. 4. Reinjection of large doses after from twelve to eighteen hours, with repetition in malignant cases, is indicated.

ROYSTER, University, Va.

CONVALESCENT SERUM IN PROPHYLAXIS OF MEASLES, CHICKEN-POX AND MUMPS. WILLIAM GUNN, Brit. M. J. **1:**183 (Jan. 30) 1932.

The author points out the difficulties in experimental work on measles, chickenpox and mumps, largely because of the fact that one must rely entirely on the history and this is often inaccurate, there being no tests of susceptibility in these diseases. Convalescent serum in measles has given the author consistently good results. When a particular serum fails to protect, this is probably due to an erroneous diagnosis of measles in the donor. All injections are given intramuscularly as soon as possible after exposure to infection. A minimum dose of 5 cc. is advisable, though smaller doses of a potent serum are effective; from the age of 3 years the dose is reckoned in cubic centimeters by multiplying the age in years by two. If given after the sixth day of the illness a modified attack usually occurs. Modified attacks of measles followed reexposure to infection on the twenty-fifth, twenty-eighth and thirty-second days when the serum had apparently protected in the first instance. Experiments were made along the same lines in an effort to immunize against chickenpox. The author concludes that, although some apparent results were obtained, convalescent chickenpox serum is far inferior to convalescent measles serum as a prophylactic agent.

Similar experiments were carried out in regard to mumps, and the author's conclusion is that the results are admittedly unsatisfactory but justify a further trial of the method with larger doses of serum.

He then discusses the Wassermann reaction during infection and states that there are two points of view on the subject. One is that scarlet fever and tuberculosis are both capable of returning a positive Wassermann reaction and possibly some other infections, although some authorities still deny that a positive Wassermann reaction is every present apart from syphilis. He then states his experiments and observations on the Wassermann reactions of the blood in measles, chickenpox and mumps. The results are not conclusive and in some respects are contradictory. "The interpretation of these changes in the Wassermann reaction is not clear, but it would appear that in the acute fevers there is an increase in the normal lipoidophile antibody content of the human serum, or a physical or chemical alteration of its constitution which permits complement-deviation to occur even in the absence of luetic infection (Mackie and Watson, 1926). This change in the serum is present only for a short time (third to twenty-first day), and rarely persists into the fourth week. Moreover, it is only found in fresh serum (first to fourth days after withdrawal) and always disappears within a week after storage at 4° C. It is not improbable that the positive reac-

tions reported in the acute fevers by various workers are attributable to the presence of this or like substance in fresh serum, and have no relation to luetic infection."

ROYSTER, University, Va.

TWO CASES OF VACCINIA. RICHARD W. TAYLOR, Brit. M. J. **1**:424 (March 5) 1932.

The author reports two cases, apparently of vaccinia, occurring in milkers of the same cow which had been ill and had an ulcerated udder.

ROYSTER, University, Va.

GLANDULAR FEVER OR INFECTIVE MONONUCLEOSIS. W. T. BENSON, Edinburgh M. J. **39**:63 (May) 1932.

The author reports six cases of glandular fever in adults and adolescents; the three youngest patients were 16, 14 and 13 years of age. He quotes Tiday and Daniel as stating that glandular fever is an acute infectious and contagious disease especially of childhood. Five of Benson's cases were sporadic. In all of the patients the lymphocytosis was absolute. The large lymphocytes were seven times as common as the small. The mononucleosis may not be present in the early febrile stage, but it becomes manifest as the fever leaves. Mononucleosis is high in sporadic cases in adults and lower in epidemic prevalence in children. Two of the patients had rash, and four had conditions of the throat. The author quotes many writers in the literature of this subject. He insists wisely that before one decides on glandular fever as a diagnosis of cervical adenitis, there should be corroborative evidence of epidemic prevalence, or the observations on the blood should be typical.

NEFF, Kansas City, Mo.

COMPLEMENT FIXATION IN MENINGOCOCCAL INFECTIONS AS AN AID TO DIAGNOSIS. H. A. COOKSON, Edinburgh M. J. **39**:258 (April) 1932.

Cookson, the pathologist at the Sunderland Royal Infirmary, shares the general opinion that in some cases meningococcal infection may be difficult to prove in the laboratory. In the septicemic stage or type, the cerebrospinal fluid may not show an increase in cells, and the cultures may not show growth. He investigated a series of serums and concludes that when no alteration can be found in the spinal fluid, examination of the blood serum by complement-fixation tests may give a clue to the nature of the condition.

In discussing this paper by Cookson, Dr. Thorp stated that it is now his routine procedure to send a specimen of blood with the first specimen of spinal fluid so that the complement-fixation test may be made. He thinks that the method is a help in making a diagnosis earlier in the disease than is often otherwise possible.

NEFF, Kansas City, Mo.

LATENT INFECTION WITH C. DIPHTHERIAE IN ASSOCIATION WITH BACTERIAL EXPERIENCE AND SCHICK IMMUNITY. SHELDON F. DUDLEY, J. Hyg. **32**: 193 (April) 1932.

Observations were made on one thousand school boys from 11½ to 15½ years of age from 1923 through 1930 to determine the relation between symptomatic diphtheria, carriers of virulent and of avirulent organisms and active immunization.

In the summer terms, symptomatic diphtheria was lowest, while the carrier rate was the highest, which coincides with Doull's observations that the warmer the climate and weather the less the incidence of diphtheria but not of the carrier rate. No constant relation between the prevalence of cases of diphtheria and carriers of virulent diphtheria bacilli was found when specific organisms were isolated in pure culture from apparently healthy boys. Not quite half of

these positive cultures were toxogenic. The mean carrier rate for toxogenic organisms was 2.9 per cent. The total rate and the relation of virulent to avirulent cultures were variable, but on the average slightly less than half of all carriers harbored the virulent form. The carrier rate for avirulent diphtheria bacilli among boys recently infected with virulent organisms was three times the expected rate, while the reverse did not occur. There is reason to believe that virulent cultures gradually become avirulent. Carriers of toxogenic organisms almost always give negative reactionns to the Schick test. Age and the grouping together of children increased immunity at a rate at which about 50 per cent of the susceptible children acquired immunity by the next year.

JANNEY, Milwaukee.

THE RÔLE OF THE HAEMOLYTIC STREPTOCOCCI IN INFECTIVE DISEASE. C. C. OKELL, Lancet **1**:761 (April 9); 815 (April 16) and 867 (April 23) 1932.

"The haphazard sequences and relationships which I have selected from an almost unlimited series, old and new, seem to constitute a *reductio ad absurdum for* any specificity view of the haemolytic streptococci and the common human diseases with which they are related. . . . The unitarian view is consonant with the admitted failure of the bacteriologist to subclassify the group. It is in consonance with the curious sequences of propagation which may be observed in streptococcal disease."

LANGMANN, New York.

MENINGITIS AND SEPTICAEMIA DUE TO MORGAN'S NO. 1 BACILLUS. ROBERT GITTINS AND J. C. HAWKSLEY, Lancet **1**:1040 (May 14) 1932.

Two cases are reported from the Birmingham Children's Hospital. In the first, a boy, aged 1½ years, died two weeks after the onset of symptoms. The spinal fluid was turbid and showed a high content of polymorphonuclear cells. Dried smears revealed a gram-negative coliform bacillus. The fermentation reactions were characteristic of Morgan's no. 1 bacillus, and agglutination tests with Morgan's antiserum gave positive reactions.

The other patient, a boy, aged 8 years, was admitted because of severe headache and vomiting of two days' duration. He had a temperature of 102.8 F. on admission and showed signs of a mild bronchitis. There was also slight stiffness of the neck and legs. Lumbar puncture revealed a clear fluid under increased pressure. A culture of the blood showed a gram-negative bacillus. The patient improved rapidly and was discharged as cured. The fermentation reactions and agglutination tests of the organism recovered from the blood were positive for Morgan's bacillus no. 1.

LANGMANN, New York.

VENTRICULAR FIBRILLATION: A RARELY RECOGNIZED CAUSE OF DEATH IN DIPHTHERIA. J. CHALIER and ROGER FROMENT, Arch. d. mal. du cœur **25**: 102 (Feb.) 1932.

Strecher's articles on "Electrocardiographic Changes in Diphtheria," published in the *American Heart Journal* in 1928 and 1929, deal with what he calls "auriculoventricular dissociation," but in this article the authors point out the relation of Strecher's findings to what they speak of as "ventricular fibrillation."

They state that the tendency of the ventricle to fibrillate manifests itself hours or days preceding death and that the prefibrillatory arrhythmic tachycardia may be the first and perhaps the only sign pointing to a fatal issue.

Reproductions of some of Strecher's electrocardiographic tracings are included in the article.

TURNER, Chicago.

RELAPSE IN TYPHOID FEVER. S. BARTSOCAS, Arch. de méd. d. enf. **35**:377 (July) 1932.

Some confusion still exists among clinicians with respect to the significance of the expressions recrudescence, relapse and recurrence in typhoid. The author

attempts first to clarify this terminology: he defines recurrence as a distinctly new and independent attack, possibly years after the initial one, in a healthy subject who has not been immunized by the first invasion. Relapses and recrudescences are more difficult to separate and are consequently often misinterpreted. A relapse may be considered, in the words of Hutinel, as "the reproduction after establishment of convalescence, in whole or in part, from the symptomatology that characterized the original attack." The period of apyrexia here is variable. Recrudescence may be defined as an aggravation of temperature and of other classic symptoms of typhoid, either during the stage of continued fever or during defervescence, but always before the establishment of apyrexia. Bartsocas presents historical data on this subject since the first thesis by Taupin in 1839; he discusses the etiology and the pathogenesis of relapse, its clinical picture and its treatment, presenting throughout the extensive monograph numerous case histories to illustrate the syndrome.

AMESSE, Denver.

TREATMENT OF PERTUSSIS WITH MIXED STREPTOCOCCUS VACCINE. M. MAKACHVILI, Nourrisson **20**:91 (March) 1932.

The good results reported are believed to be due to nonspecific protein therapy. The vaccine was made from strains of streptococci obtained from cases of scarlet fever. The supernatant fluid was decanted and replaced with dealbuminized toxin according to the technic of Nipton. The local and general reactions were mild.

MCCLELLAND, Cleveland.

ANTIDIPHTHERITIC VACCINATION WITH ANATOXIN. G. RAMON AND R. DEBRÉ, Presse méd. **40**:545 (April 8) 1932.

Immunization against diphtheria by means of injection of anatoxin was proposed eight years ago, and has come into general use in most countries. The safety of this method has been amply proved, even with adults and tuberculous patients, for whom some physicians have claimed that a contraindication exists. The method is efficacious to a high degree, producing in 80 per cent of cases a much higher antitoxin titer in the blood than is necessary for complete protection.

Recently the French army adopted "associated vaccination," and since mixing any one vaccine with another increases the possibility of reactions, anatoxin would appear to be innocuous, even in adults.

Experiments with purified or extracted anatoxin have given no more satisfactory results than the ordinary anatoxin. Various methods of administration (oral, nasal and percutaneous) have demonstrated the vast superiority of the injection method. This method seems to confer the maximum immunity for the greatest number of persons, with a minimum of inconvenience.

BIEKER, St. Petersburg, Fla.

THE PRACTICE OF COMBINED TYPHOID-PARATYPHOID-DIPHTHERIA VACCINATION. J. FRICKER AND L. FERRABOUC, Presse méd. **40**:873 (June 1) 1932.

In 1926 Ramon and Zoller introduced a combined typhoid-paratyphoid-diphtheria vaccination, producing a rapid reversal of a positive Schick reaction. This they termed "associated vaccination." In 1930 this was adopted by the French army, being reserved particularly for units exposed to diphtheria. Three injections are given: the first of 1 cc. of typhoid-paratyphoid A and B, anatoxin 0.5 cc.; the second of 1 cc. of typhoid-paratyphoid A and B, anatoxin 1 cc., and the third of anatoxin alone, 5 cc. The first two injections are given eighteen days apart, and the second and third, fifteen days apart. The vaccines are mixed at the moment of injection. Contraindications are acute febrile conditions and renal insufficiency. Even a transient albuminuria may indicate a state of "renal debility" which elimination of bacterial proteins might aggravate, and should

constitute a contraindication. The subjects are given complete rest for twenty-four hours on a milk diet.

The authors have made 1,110 associated vaccinations with very satisfactory results. Reactions have been moderate. No case of acute nephritis with hematuria has been observed. One patient had anuria and vomiting. The usual reaction is a local one, with pain and erythema persisting for not more than three days, and a general reaction with fever lasting for perhaps forty-eight hours. The second injection produces milder reactions, and the third none at all. It is possible that the number of injections may be reduced to two by using anatoxin of increased antigenic value, as suggested by Ramon and Nelis.

The method seems to be particularly easy and safe for rapid immunization against both diphtheria and typhoid. BIEKER, St. Petersburg, Fla.

BLOOD LACTIC ACID IN ACUTE INFECTIOUS DISEASES. E. LIVERANI, Arch. di farmacol. sper. (no. 11) **51**:245 (Feb. 20) 1931.

Blood lactic acid was determined in fifty-six cases of acute infectious diseases by the method of Furth and Parnas, as modified by Clausen and by Folin and Wu. The blood lactic acid was always abnormally high, especially in those cases in which there was pronounced toxemia. This increase in lactic acid, when there were no factors present affecting the normal functioning of the cardiac and respiratory apparatus, was attributed to the following causes: increase in the general metabolism, functional hepatic insufficiency, deficient tissue oxygenation and increased fermentative activity in the intestine. KUGELMASS, New York.

THE INFLUENCE OF MALARIA IN THE MOTHER ON THE PHYSICAL DEVELOPMENT OF THE CHILD IN THE FIRST YEAR OF LIFE. A. MARTIGNETTI, Riv. di clin. pediat. **30**:11 (Jan.) 1932.

The author compared 240 children of mothers who had malaria before or during pregnancy and a similar group of 240 children of nonmalarial mothers. There were 20 babies 1 month of age, and 20 for each succeeding month up to 1 year of age. The families were of the average social status from the district around Cagliari. Only healthy children were used.

The children of malarial mothers averaged about 400 Gm. lighter in weight during the first seven months; later the difference was about 250 Gm. Similarly, the children of malarial mothers were definitely shorter (about 2 cm.) when about 7 months old, but less than 1 cm. shorter at 1 year. The children of the malarial mothers tended to get their teeth later and to walk later.

One hundred and seventy-one of the malarial mothers and 186 of the control mothers had sufficient milk to nurse their babies. There was a higher percentage of abortions and of premature births among the malarial mothers.

HIGGINS, Boston.

PERSISTENCE OF ANTIBODIES (DIPHTHERITIC ANTITOXIN) IN ANAPHYLACTIC SENSITIZATION. A. FOA, Riv. di clin. pediat. **30**:59 (Jan.) 1932.

Two groups of guinea-pigs were studied. One group had been given horse serum from eight to fourteen days previously. The animals were given a lethal dose of diphtheria toxin; this was followed at intervals of from twelve to one hundred and ninety-two hours by antitoxin. The animals that were previously sensitized died sooner than the normal controls. Therefore sensitization of the animals had diminished their antitoxin. The loss of antitoxin was from the tissues quite as much as from the blood. HIGGINS, Boston.

POSTDIPHTHERITIC HEMORRHAGE DUE TO CEREBRAL EMBOLISM AND CARDIAC THROMBOSIS. EUGENIA S. YANNUZZI, B. ELGADO CORREA AND A. VOLPE, Arch. de pediat. d. Uruguay **3**:75 (Feb.) 1932.

This complication, although rare in diphtheria, occurred in a child 6 years of age affected with a pharyngeal diphtheria. One hundred and fifty cubic centimeters

of antidiphtheritic serum was given. On the third day after the development of the disease, paralysis of the face and of the left arm and leg was noticed. The condition was probably caused by an embolus of cardiac origin.

The child died a few days later, showing symptoms of intense dyspnea, extreme pallor and convulsions. This condition is fortunately one of the rarest complications of diphtheria.

SCHLUTZ, Chicago.

SERUM THERAPY IN SCARLET FEVER. WERNER LOOK, Arch. f. Kinderh. **96**:129 (March 6) 1932.

The result obtained with various types of serum therapy (at the Krankenhauser Charlottenburg Weslend) in scarlet fever from 1925 to 1931 are tabulated and discussed. Of 158 patients, 134 were treated with the ordinary *Sharlach Heilserum der Behring werke, Marburg,* 2 with a concentrated preparation of that serum, 9 with the concentrated serum of Schering-Kahlbaum, 3 with the combined *Sharlach Streptokokken Serum, Hoechst,* 3 with normal human serum and 7 with human convalescent serum. In 4 cases blood transfusions were given, the donors being either persons convalescent from scarlet fever or healthy adults. Except in the first group, the numbers are so small that no definite conclusions can be drawn as to the benefit derived. In the first group, 75.4 per cent showed a definite drop in temperature and general improvement. The decrease in complication was not, however, as great as that reported by Prinzing following the use of normal and convalescent human serum. The results were better when the serum was given early in the disease. Blood transfusion would seem to offer some benefit when it is impossible to obtain serum or when contraindications to the use of serum exist.

LESLIE, Evanston, Ill.

CARDIAC LESIONS IN SCARLET FEVER, STREPTOCOCCUS INFECTIONS AND RHEUMATIC GRANULOMATOSIS. T. FAHR, Beitr. z. path. Anat. u. z. allg. Path. **85**:445, 1931.

The focal and diffuse histiocytic proliferations in the heart first described by Fahr in scarlet fever are regarded by him as allergic reactions to the streptococci usually associated with scarlet fever. These changes are, however, in marked contrast to the Aschoff nodule in rheumatic fever. This nodule is as specific a granuloma as the lesions of tuberculosis or of Hodgkin's disease.

BOIKAN, Chicago. [ARCH. PATH.]

A CASE OF PERTUSSIS ENCEPHALITIS IN A SYPHILITIC CHILD. W. MIKULOWSKI, Jahrb. f. Kinderh. **135**:207 (April) 1932.

A child, 2 years old, who had had an attack of pertussis at 1 year, one week before admission to the hospital, had generalized edema, vomiting and cough. Examination showed hyptonia, nystagmus, staggering gait, a masklike face and somnolence; the Wassermann reaction of the blood was positive; the spinal fluid was normal. The respiratory symptoms became typical of pertussis pneumonia, after which the patient recovered completely.

CLAUSEN, Rochester, N. Y.

SEROUS PLEURISY OCCURRING IN THE COURSE OF INFECTIOUS DISEASES IN CHILDHOOD. H. ZISCHINSKY, Jahrb. f. Kinderh. **135**:213 (April) 1932

Forty cases observed during one year at Pospischill's clinic showed moderate fever, few symptoms and clear fluid in small amount, demonstrated by physical signs, spinal puncture and roentgen examination. There was rapid recovery.

CLAUSEN, Rochester, N. Y.

EPIDEMIOLOGY OF SCARLET FEVER IN DÜSSELDORF FROM 1901 TO 1930. R. GAUS, Jahrb. f. Kinderh. **135**:220 (April) 1932.

Three major epidemics of scarlet fever occurred in Düsseldorf, ten years apart. A minor increase of the disease occurred five years before the first major epidemic. The major epidemics lasted two or three years. The morbidity has steadily declined, as has the mortality, which fell in the last epidemic to less than 1 per cent (1927 to 1930). Children are most frequently attacked between the ages of 5 and 10 years, less between 1 and 5 years and least between 10 and 15 years. Infants are rarely attacked. The greatest mortality is between the ages of 1 and 5 years. Between 1925 and 1930, 0.25 per cent of the entire population and 9.4 per cent of children between 5 and 10 years of age contracted scarlet fever. Rich and poor are affected alike. During the months with the highest temperatures (July and August), there is least scarlet fever; during those with the lowest temperatures, there is on the whole the most scarlet fever.

CLAUSEN, Rochester, N. Y.

FURTHER EXPERIENCES WITH PERTUSSIS VACCINE. H. SCHMIDT and M. WEICHSEL, Monatschr. f. Kinderh. **53**:373, 1932.

In twenty cases of pertussis treated with a certain vaccine, labeled I. G., results were disappointing both for the active treatment and for prophylaxis.

GERSTLEY, Chicago.

THE TREATMENT OF PERTUSSIS WITH I. G. VACCINE. BAUMLER, Monatschr. f. Kinderh. **53**:379, 1932.

The treatment of pertussis with I. G. vaccine, both prophylactic and active, is unreliable.

GERSTLEY, Chicago.

THE PROGNOSIS OF DIPHTHERITIC INVOLVEMENT OF THE HEART AND CIRCULATION. F. THOENES, Monatschr. f. Kinderh. **53**:381, 1932.

During the years 1929 and 1930, the writer studied one hundred and twenty-eight patients with diphtheria, of whom ninety-five showed definite cardiovascular complications. Of these ninety-five, examination from nine to thirty-three months later showed only seventeen to have any objective abnormal cardiac findings, while another sixteen complained of cardiac disturbance, but nothing organic could be diagnosed.

The mortality of one hundred and eighty-six children with diphtheritic complications was 31.3 per cent.

Chronic cardiac complication is perhaps a little more inclined to develop in patients showing complication early than in patients without this complication, but the difference is so slight that it cannot be used in prognosis. Again there is no symptom, not even the much feared "gallop rhythm," that can be relied on as an aid to prognosis.

The treatment is, of course, prolonged rest in bed.

GERSTLEY, Chicago.

TREATMENT OF TETANUS. VILEM VEZER, Časop. lék. česk. **69**:1164 (Aug. 8) 1930.

The author reports four cases in children, in three of which there was an incubation period of twenty days. All of the patients had severe typical convulsions and trismus. In addition to the usual symptomatic treatment and the repeated spinal punctures made with the patient under ether anestheia, on the first two days one fourth of the spinal fluid was replaced by antitoxin. After that from one-fourth to one-half of the fluid was replaced by a 15 per cent solution of magnesium sulphate until a ten hour magnesium palsy replaced the convulsion.

The intravenous administration of magnesium sulphate, twice a day in doses of 10 cc. was continued until at least two days had elapsed without the occurrence of retrogressive automatic movements. Rectal instillations of chloral hydrate, phenobarbital and sodium bicarbonate were given until after ether anesthesia had been produced and lumbar puncture performed and until automatic movements had been replaced by voluntary movements. The four patients made a complete recovery.

STULIK, Chicago.

FIGHTING INFECTIOUS DISEASES WITH VACCINATION. H. BROKMAN, Pediatria Polska **2**:300 (May) 1931.

The author discusses the merits of vaccination pro and con. Much research has to be done along this line. A great stride has been taken in scarlet fever and diphtheria immunization; however, little is known about German measles, chickenpox and smallpox. At present vaccination is the only logical method that one can pursue until something more positive is found.

KRUPINSKI, Chicago.

POSTDIPHTHERITIC CHANGES IN THE EYE. K. G. PLOMAN, Acta ophth. **9**:221, 1931.

Paresis or paralysis of accommodation is a common postdiphtheritic disturbance of the ocular muscles. Changes in the pupil, on the other hand, are rare. Reflected rigidity of the pupils (loss of the pupil contraction in near focusing when the power of convergence is retained and the reaction to light is normal) has not been observed. Unilateral or bilateral ptosis of the upper eyelid is an unusual symptom. Paresis of the abducens occurs comparatively frequently, whereas paresis of the trochlearis is practically unknown. The effect may manifest itself at any point of the nerve passage, from the center to the periphery and at times in the muscle itself. This paper has an excellent survey of the literature on ocular disturbance in diphtheria.

YUDKIN, New Haven, Conn. [ARCH. OPHTH.]

SEASONAL VARIATIONS IN DIPHTHERIA MORTALITY. P. H. ANDERSON, Ugesk. f. læger **94**:335 (March 31) 1932.

There was found a distinct increase in mortality from diphtheria during the months of March, April and May, based on the reports of the health department and the records of the Municipal Contagious Disease Hospital.

ELGHAMMER, Chicago.

PANCREATITIS AS A COMPLICATION OF PAROTITIS EPIDEMICA. EJNAR SYLVEST, Ugesk. f. læger **94**:508 (May 19) 1932.

The author relates a number of cases of pancreatitis occurring in an epidemic of parotitis.

ELGHAMMER, Chicago.

Chronic Infections

AN INVESTIGATION OF SPIROCHETOSIS OF THE DENTAL ANLAGE IN CONGENITAL SYPHILIS. THOMAS J. HILL, Am. J. Path. **7**:515 (Sept.) 1931.

The enamel organ of the central incisor and first molar of the permanent teeth was examined by various spirochete stains. Despite the demonstration of spirochetes in six livers from cases of congenital syphilis, it was impossible to find them in the dental anlage. Therefore, it is concluded that the characteristic congenital syphilitic dental deformities are not the direct result of the invasion of the enamel organ by Spirochaeta pallida.

MOORE, Charlotte, N. C.

CHILDHOOD TUBERCULOSIS: A MISNOMER. ARTHUR J. BLAIR and LOUIS ROSENBAUM, M. J. & Rec. **136**:19 (July 6) 1932.

There seems to be a diversity of opinion in the minds of the members of the medical profession as to what childhood tuberculosis actually implies. The conception entertained by many, that a positive reaction to tuberculin and roentgen evidences of increased hilar markings with calcification of some of the tracheobronchial glands are the basic factors on which a diagnosis is to be made, seems to be erroneous. These findings are indicative of, and represent the sequence of events of, a primary tuberculous infection of the lungs which is present in a number of children, depending on age, exposure and other factors and should be designated as a primary pulmonary tuberculous complex. Tuberculosis in children, like tuberculosis in the adult, to the minds of the authors, represents a definite clinical and pathologic entity, with tuberculous involvement of the parenchyma of the lung.

WILLIAMSON, New Orleans.

THE SANATORIUM CHILD. LEON A. ALLEY, New England J. Med. **207**:884 (Nov. 17) 1932.

During the past five years 178 children were admitted to the Lakeville Sanatorium who were in their first decade of life. The diagnosis of tuberculosis had been made in every case. In the majority of cases the involvement was in the bony structures of the body. Of these children 169 were white and 9 were Negroes. In 19 of these cases the condition was eventually proved to be nontuberculous. Eleven children died of tuberculosis in the sanatorium.

The original source of infection was either human sputum or cow's milk. Sixty-eight gave a history of other cases of tuberculosis in the family. Careful study of a series of 100 cases in the sanatorium showed that in 4 per cent of the patients under 10 years of age with tuberculosis of the lymph glands and in 16 per cent of those with tuberculosis of the bones and joints in the same age group the condition was due to infection with the bovine type of organism. By the Pirquet method, the percentage of positive reactions was as follows: human, 7.75 per cent; bovine, 8.25 per cent, and both human and bovine, 84 per cent. No positive reactions were obtained by the Mantoux method in the cases in which the reaction to the Pirquet test was negative.

Observations show that weight below normal is not found more frequently in children who react to tuberculin than in those who do not. Treatment in a sanatorium or its equivalent should be given children who show: (1) tuberculous lesions accompanied by symptoms and physical signs referable to tuberculosis; (2) latent lesions of the lung of childhood or adult type that on repeated examinations are found to be progressive; (3) massive tuberculosis of tracheobronchial lymph nodes that has not undergone calcification.

Regardless of the disease, rest is the greatest natural medical ally. It is useless to coax or to try to force a child to eat. Cod liver oil has a distinct value as a food. Out-of-door air should be used generously day and night, winter and summer. While in the open air the rays of the sun judiciously used have a very beneficial effect on the human body. Artificial heliotherapy indoors is but a substitute at best.

GENGENBACH, Denver.

TUBERCULOUS DISEASE IN CHILDREN: ITS PATHOLOGY AND BACTERIOLOGY. JOHN W. S. BLACKLOCK, Privy Council, Medical Research Council, Special Report Series no. 172, 1932.

This investigation is based on the autopsy findings in 283 cases showing tuberculous infection from a series of 1,800 consecutive autopsies performed by the author at the Royal Hospital for Sick Children, Glasgow, from March, 1924, to November, 1931. The tubercle bacilli were isolated from 183 cases of the 216 in which it was attempted. Of 65 children with surgical tuberculosis (no deaths), the organism was isolated from 52 (either by direct culture or by guinea-pig inoculation). There were 152 human strains and 89 bovine.

The primary infection was considered to be thoracic in 61.1 per cent and in these was predominantly due to human strains, while in 35.7 per cent of the cases the primary focus was abdominal and chiefly due to bovine types of bacilli. The methods used in culturing and typing the organisms are explained in detail. The gross and microscopic pathologic process in tuberculosis in infancy and childhood is fully described. The author correlates his findings and discusses many of the debated problems of this disease with numerous references to the work of other men. This thorough and extensive study furnishes a valuable addition to the literature on this subject. LEMASTER, Evanston, Ill.

INTRATHORACIC CALCIFICATIONS IN CHILDHOOD. M. PÉHU and M. LAMBERT, Rev. franç. de pédiat. **8**:401, 1932.

This excellent article refers to calcium deposits in the lungs, pleura and glands of the thorax produced by the tubercle bacillus. The authors report the cases of fourteen boys and fourteen girls, the ages ranging between 4 and 15 years, the predominance being in early childhood. The children were examined because of general symptoms in ten cases, pulmonary symptoms in six cases, by chance in three cases, and for various causes in the remainder. Only two cases showed the bacilli; thirteen patients had a positive skin reaction and three a negative reaction. The remainder were not tested. Two patients died, both at 11 years, one from a fibrocaseous development and the other from meningitis. The only diagnostic sign is the roentgenogram. Eight illustrations are shown. The differential diagnosis lies between enlarged, noncalcified glands, images of vessels and foreign bodies. Two less common errors may be made by considering injections of iodized poppy seed oil 40 per cent, which produce an iodide base, and pneumonoconiosis. A complete bibliography is given. SANFORD, Chicago.

A CONTRIBUTION TO THE STUDY OF CHRONIC MILIARY TUBERCULOSIS IN INFANCY. A. VALLETTE and B. ROSENKRANZ, Rev. franç. de pédiat. **8**: 462, 1932.

The authors present the cases of three children, 12, 8 and 5 years of age, respectively, who clinically had tuberculosis, and by roentgenogram showed mottling of the lung with diffuse granulations. No particular change was observed in the thirteen months that they were in the hospital under observation. The prognosis is uncertain; one patient remained the same; one died of tuberculous meningitis, and the third seemed to improve. The treatment appears to be hygienic and dietetic. SANFORD, Chicago.

METHOD OF AMPLIFYING THE CUTANEOUS REACTIONS OF TUBERCULIN AND OF PREVENTING SYSTEMIC REACTIONS. A. BOUVEYRON, Rev. de la tuberc. **13**:603 (June) 1932.

The addition of a solution of epinephrine to tuberculin in the performance of the Pirquet test serves to augment the local cutaneous reaction, even when weaker dilutions of tuberculin are employed. This technic also minimizes the likelihood of general reactions in highly allergic persons. The author prefers one of the two following procedures: 1. To the scarification apply a 1:100 or 1:400 solution of epinephrine, and allow it to remain until blanching occurs. Dry and apply one drop of a mixture of epinephrine and tuberculin (1:3), and allow it to remain from fifteen to twenty minutes. 2. This differs from the first method only in that after the preliminary application of epinephrine tuberculin alone is applied to the scarification. The augmented local reaction appears to be due to the fact that during the entire phase of local vasomotor constriction the scarified surface is exposed to a high degree of concentration of tuberculin, since diffusion and admixture with tissue fluids are inhibited. This technic reduces to the minimum the absorption of tuberculin and at the same time amplifies the local cutaneous response to this agent. SMITH, Ogden, Utah.

AN ABNORMAL ALBUMIN-CELL RATIO IN THE SPINAL FLUID OF A PATIENT WITH TUBERCULOUS LEPTOMENINGITIS. G. SALVIOLI, Scritti med. dedicati a Carlo Comba, ediz. Riv. di clin. pediat., 1929, p. 793.

The abnormal albumin-cell ratio of Sicard and Foix is a condition in the spinal fluid when the cell count is low or normal and the albumin is high. It has been reported in: (1) compression of the spinal cord, (2) cerebral hemorrhage, (3) early stages of cerebral softening, (4) poliomyelitis, (5) necrotic myelitis, (6) optic neuromyelitis and (7) cerebral compression.

The author reports the case of a tuberculous child of 2 years, who had solitary tubercles of the meninges. The spinal fluid showed the abnormal albumin-cell ratio of Sicard and Foix, adding another condition to those in which this finding occurs.

HIGGINS, Boston.

VACCINATION AGAINST TUBERCULOSIS BY THE METHOD OF CALMETTE-GUÉRIN. JUAN L. GIRON, Arch. d. hosp. de niños Roberto del Rio **3**:40 (March) 1932.

Bacillus Calmette-Guérin given by the oral route is absorbed by the intestine of the child. The allergic manifestation following the oral vaccination occurs late and is quite variable. The vaccine given in the usual dose (3 cg.) seems to be entirely harmless to the child.

To use the method incompletely or not according to the exact direction of the original authors will almost certainly lead to failures. The author is certain that the method is harmless if properly applied, but believes that insufficient data are as yet at hand for one to be certain of its efficacy.

SCHLUTZ, Chicago.

DIAGNOSIS OF SYPHILIS BY MEANS OF A DESICCATED AND DEFIBRINATED DROP OF BLOOD. ALEJANDRO CHEDIAK, Arch. de med. infant. **1**:3 (Feb.) 1932.

The author has used this method in 254 cases. In these cases 49 positive reactions were obtained by a Wassermann test, 58 by a Kahn test, and 48 with the drop method. This method, therefore, closely approximates the results obtained by the other more elaborate tests. The blood is drawn from the lobe of the ear or from the toe. A drop is placed on a slide and defibrinated with a pinpoint. The remainder of the method is a process of complement and flocculation. The advantages are that it dispenses with venipuncture, which is difficult in infants and fat persons, and that in urgent cases, such as those in which a transfusion is to be given, this method gives an idea of the serologic state of the donor.

SANFORD, Chicago.

LOCALIZED TUBERCULOSIS OF THE CHILD "LOBITIS TUBERCULOSA." PEDRO CANTONNET BLANCH, Arch. pediat. del Uruguay **3**:293 (July) 1932.

This condition, which was considered characteristic only of adult tuberculosis, is found more frequently in children than was formerly believed. Tuberculous lobitis can be an acute, subacute or chronic process, depending on the type of lesion and the stage of infection. Two forms of tuberculous lobitis are commonly recognized: a curable form, the so-called epituberculosis, and an evolutionary form which may take one of three courses, namely, caseation, ulceration or sclerosis. Of these forms, only the sclerotic form is favorable as regards prognosis.

In the author's experience, pneumothorax is the most effective form of treatment if an active tuberculous focus is present.

SCHLUTZ, Chicago.

HOOK NEEDLE FOOT A SIGN OF CONGENITAL SYPHILIS. M. TORROELLA, Gac. méd. de México **63**:353 (July) 1932.

The author describes a deformity of the feet in the new-born which consists in extreme flexion of the foot over the leg, giving the extremity the appearance of a hooked knitting needle. This condition seems to be due to a preponderance of

the action of the flexor muscles of the leg. In some cases, the hypotonia is so extreme that the dorsum of the foot may be brought in contact with the anterior surface of the leg.

PARDO-CASTELLO, Havana, Cuba. [ARCH. DERMAT. & SYPH.]

DYSTROPHIES OF THE SKULL AND AORTIC ENLARGEMENT IN CONGENITAL SYPHILIS. BENITO SORIA, Rev. méd. latino-am. **17**:1359 (July) 1932.

The author has observed the frequent association of deformities of the skull and enlargement of the aorta in congenital syphilis. He contends that in all children with dystrophic cranial deformities a roentgenographic study of the aorta should be made. The diameter of the ascending aorta in normal children from 2 to 8 years of age is 1 cm. as a maximum, and in children from 9 to 14 years old it should not be more than 1.3 cm. Higher figures are considered pathologic. Eight cases are reported in detail.

PARDO-CASTELLO, Havana, Cuba. [ARCH. DERMAT. & SYPH.]

A RARE FORM OF SYPHILITIC OSTEOCHONDRITIS. MARTHA KLEIN, Arch. f. Kinderh. **95**:242 (Feb. 5) 1932.

Excellent roentograms are shown of a patient with severe osteochondritis corresponding to the active type of Schneider, in which there is an extensive growth of granulation tissue into the epiphyseal-diaphyseal border, giving a double contour to the bones as seen in the roentgenogram. This characteristic was particularly striking in the bones of the pelvis. There is a good discussion of the underlying pathologic anatomy.

LESLIE, Evanston, Ill.

SEROLOGIC TESTS FOR SYPHILIS IN A CHILDREN'S HOSPITAL. E. ROMINGER and L. SZEGO, Arch. f. Kinderh. **95**:256 (Feb. 5) 1932.

Wassermann and Sachs-Georgi tests have been made on all patients entering the Universitäts Kinderklinik at Kiel, since October, 1926. Up to the end of 1930, 4,658 tests were made. Positive reactions were obtained in 115 of 117 definitely established cases of syphilis. Nonsyphilitic reactions were obtained in nine cases; in five of these cases the condition was scarlet fever and in the sixth, hepatitis; the seventh case was that of a 7 months old baby with pneumonia; the eighth, that of a 2 year old girl with otitis media and cervical adenitis, and the ninth, that of a 12 year old girl with diabetes. The authors advocate serologic tests for syphilis as a routine, because they believe that without such tests many cases would have been missed. Their experience with new-born infants was limited, so that they do not feel qualified to speak on the reliability of serologic tests made at this age period.

LESLIE, Evanston, Ill.

THE RELATION BETWEEN MENINGITIS AND THE STAGES OF TUBERCULOSIS IN REGARD TO TIME AND PATHOGENESIS. D. OROSZ, Arch. f. Kinderh. **97**: 216 (Oct. 4) 1932.

Investigation showed that tuberculous meningitis most frequently occurs from two to three months after the first infection, and usually within from five to six months. The author believes that the onset of the meningitis corresponds to the early secondary stage of tuberculosis. During the late secondary stage, the incidence of meningitis is greatly reduced and is definitely related to such intercurrent factors as measles and pertussis. Meningitis rarely occurs during the primary or tertiary stage of tuberculosis.

LEMASTER, Evanston, Ill.

THE DEVELOPMENT OF CONGENITAL SYPHILITIC CHILDREN AND HEALTHY CHILDREN OF SYPHILITIC MOTHERS. FRIEDRICH LEO, Dermat. Wchnschr. **94**:787 (June 4) 1932.

This is a detailed statistical study of the development of children with congenital syphilis observed between 1908 and 1930. Of 175,108 children seen,

0.31 per cent were syphilitic; 56 per cent of the babies of syphilitic mothers with positive Wassermann reactions at the time of parturition remained healthy, while 76 per cent of those of syphilitic mothers whose Wassermann reaction was negative remained well. This indicates that the more thorough the treatment of the mother during pregnancy the better the chance for healthy offspring.

TAUSSIG, San Francisco. [ARCH. DERMAT. & SYPH.]

TUBERCULOSIS OF THE EXTERNAL GENITALIA FROM CONTACT INFECTION. H. VIETHEN, Dermat. Wchnschr. **95**:1469 (Oct. 8) 1932.

Two cases are described in which tuberculous ulceration occurred apparently as a result of external infection. In a boy, 10 years of age, tuberculosis of the prepuce developed secondary to a draining tuberculous inguinal gland. A girl, 15 years of age, presented a tuberculous ulceration extending from the labia onto the buttock. She had tuberculosis of the urinary tract.

TAUSSIG, San Francisco. [ARCH. DERMAT. & SYPH.]

TERTIARY SYPHILIS OF THE LIVER IN CHILDREN AND ITS RELATION TO CIRRHOSIS. U. PETERSEN, Frankfurt. Ztschr. f. Path. **43**:44, 1932.

The clinical diagnosis of the condition of a 4 year old girl was syphilitic cirrhosis of the liver. The postmortem diagnosis was lobulated syphilitic liver, enlarged spleen, lipoid nephrosis, ascites and purulent peritonitis. Even though no spirochetes were found in the liver, the diagnosis of lobulated syphilitic liver was made because of the coarse granular appearance, a productive inflammation starting from the capsule and extending into the periportal spaces, while the liver tissue in general was well preserved. Also, the lipoid nephrosis seemed consistent with syphilis. Clinically, there was a typical saddle nose, and the Wassermann reaction was positive when the child was first seen. In reviewing the literature critically, the author did not become convinced of the existence of a true syphilitic cirrhosis.

SAPHIR, Chicago. [ARCH. PATH.]

DYNAMICS OF THE SEDIMENTATION REACTION IN THE TUBERCULOUS CHILD. LADISLAUS VON CSÖKE, Jahrb. f. Kinderh. **131**:216 (April) 1931.

In 329 children with tuberculosis treated at the Johannes Krankenhaus of Budapest, the rate of sedimentation of the red cells was determined by Westergren's method. Rapid rates, in general, were found characteristic of the severe, active disease, but more characteristic was a rapid fall in the first fifteen minutes. An early slow fall was found in mild cases, and a slow, steady fall in the terminal stages of tuberculosis.

CLAUSEN, Rochester, N. Y.

STIGMAS OF CONGENITAL SYPHILIS IN THE BONES. WALTER GRAVINGHOFF, Jahrb. f. Kinderh. **133**:189 (Oct.) 1931.

The author describes roentgen changes: shadows in the spongiosa of the long bones, fine shadows parallel to the cortex, a double border about the bones of the foot, especially the calcaneum, and lines along the iliac crest. These were rarely found in children of mothers free from syphilis, but were frequent in those of syphilitic mothers, even when the mothers had received antisyphilitic treatment and when the infants showed no other manifestations of syphilis. Discussion is extremely detailed, and references to the recent literature are full.

CLAUSEN, Rochester, N. Y.

EPITUBERCULOUS PULMONARY INFILTRATION IN CHILDHOOD. WOLDEMAR BLACHER, Jahrb. f. Kinderh. **135**:1 (March) 1932.

This article consists of a critical discussion of the literature and a presentation of cases. Epituberculous infiltration in the upper pulmonary fields was chronic

in three cases; in seven cases involving middle, lower or total fields of the lungs, the course was acute. The immunobiologic reaction, as expressed in fever and by the blood picture, was poor in the younger children. Improvement was slow in the younger patients and in those with involvement in the upper pulmonary fields. Recurrence of infiltration occurred in three cases, and was mild and brief. At the height of the involvement, the percussion note was dull, and boardlike resistance was felt; breath sounds were absent; the mediastinum was displaced toward the affected side. Abnormal signs persisted for one or two years or more. At the height of the infiltration, the blood picture usually showed an improvement, in that lymphocytes increased. Signs of regression appeared in younger children in from three to eight months; in older children, in three weeks. The pleura may be involved some time in advance of involvement of the parenchyma. It was possible to observe the development of epituberculous infiltration about a known primary focus. Especially marked infiltration occurred in only 6.7 per cent of cases of tuberculosis in childhood. A constitutional predisposing factor must therefore be involved; Blacher believes that the exudative diathesis or the tendency for allergic reaction to develop may be an important factor.

CLAUSEN, Rochester, N. Y.

POLYCYTHEMIA IN GENERALIZED TUBERCULOSIS; ITS SUPPOSED DEPENDENCE ON THE SPLEEN. KARL GERHARD HEYMANN and R. BUSSEL, Jahrb. f. Kinderh. **136**:26 (June) 1932.

Polycythemia in generalized tuberculosis is not due to tuberculosis of the spleen. A critical analysis of the cases is made.

CLAUSEN, Rochester, N. Y.

CONGENITAL SYPHILIS OF THE INTESTINE. D. Y. KU, Virchows Arch. f. path. Anat. **280**:852, 1931.

Ku reports four cases of congenital syphilis with syphilitic involvement of the small intestine. He describes three histopathologic types of lesion. In one the mucosa was atrophic and was replaced by granulation tissue. Miliary syphilomas were present in the muscle coat. In a second case there was diffuse thickening of the wall owing to proliferation of the connective tissue of the mucosa and submucosa. In a third form, necrotic gummatous lesions of the mucosa and submucosa were associated with ulcers. Spirochetes were found in the tissues of the intestinal lesions in two of the cases.

SCHLUTZ, Chicago. [ARCH. PATH.]

INTRALUMBAR TUBERCULIN THERAPY OF BASILAR MENINGITIS. OTTO SAXL and ADOLF HOCHWALD, Časop. lék. česk. **70**:955 (July 3) 1931.

Cramer and Bickel regard only 60 of the 250 cases of basilar meningitis in which the condition was reported to be healed, as definitely diagnosed. Tendency to heal is greater in the young. The intralumbar injection of tuberculin was tried in eight children with basilar meningitis. In two the condition of the sensorium cleared up and the course was extremely prolonged; one boy was able to read daily for three weeks and another had a period of two months with no symptoms. However, all eight patients died. Two died within twenty-four hours, possibly from the severity of the reaction. In two cases a change from a negative Pirquet reaction to a positive one occurred. It is possible that the child with the two month remission died of reinfection or intercurrent infection. The mode and path of infection are discussed; in the case cited in minute detail, foci were found in the tonsil, which might point to a lymphatic route. The variation in the immunity factors of the spinal fluid and the blood in discussed.

STULIK, Chicago.

ATTEMPTS AT TREATMENT OF TUBERCULOUS MENINGITIS. JAROSLAV PROCHAZKA, Časop. lék. česk. **70**:1045 (July 24) 1931.

The author reviews the various treatments suggested for tuberculous meningitis and classes them as medicinal, both local and general, lumbar puncture and drainage of the spinal canal, surgical procedures and intralumbar medication. Of one hundred and fifty patients coming to the clinic or hospital, not a single one recovered. There is a possibility of localized tuberculosis of the meninges and the cases in which healing was reported may belong to this group. The diagnosis of the condition in some of the cases in which healing has been reported may be questioned. Basilar meningitis is essentially a part of a miliary tuberculosis, and in the treatment this fact should be considered.

STULIK, Chicago.

THE SO-CALLED CLOSED TUBERCULOUS PROCESS IN INFANTS AND EARLY CHILDHOOD. M. PEKERT, Orvosi hetil. **76**:534 (June) 1932.

The early and reliable diagnosis of tuberculous processes in children is extremely important. Physical findings and roentgen studies are usually of great help, but with negative results tuberculosis cannot be ruled out definitely. For this reason, examination of the sputum seems to be important and is sometimes very useful. Of course, sputum cannot be obtained from infants and young children. To obviate this difficulty, the contents of the stomach before the first meal in the morning were examined. Eighty-four children between the ages of 2 months and 3 years were thus studied. Proper control examinations were also performed. Of the cases in which the findings were positive, thirty-three were in complete harmony with the physical and roentgenologic findings; in ten cases, they were positive, even though the physical and roentgenologic findings were only suggestive, and in fourteen cases, the acid-fast organism could be found in the contents of the stomach even though the clinical and roentgen diagnosis was negative. The organisms were identified by inoculation into animals, and every possible source of error was excluded. The author concludes that the safest procedure is to consider every form of tuberculosis in infants and children as an open process.

ZENTAY, St. Louis.

THE WASSERMANN REACTION OF HUMAN MILK. K. OSHIDA, J. Pediat., Tokyo, no. 389, Oct. 20, 1932, p. 1892.

The author studied the Wassermann reaction of human milk at various stages, both before and after parturition, in ninety-seven patients admitted to the maternity ward of the Tokyo Red Cross Hospital. The findings are as follows: (1) The colostrum frequently gives a positive test when the blood is negative; (2) the transitional milk less frequently gives positive results than the colostrum, when the Wassermann reaction of the blood is negative; (3) the mature milk gives negative tests when the blood is negative; (4) a positive Wassermann reaction of the milk in patients whose blood gives a negative reaction is always transitory, and the milk in such cases is yellowish and viscous; (5) the two breasts may give similar results or may show opposite ones; (6) the Wassermann test of human milk may change from day to day; (7) the witch's milk is always negative, even though the blood is positive; (8) there is no parallelism between the Wassermann test of the blood and that of the breast milk.

KATO, Chicago.

Internal Diseases

THE RELATIONSHIP BETWEEN STATUS LYMPHATICUS, THE THYMUS AND THE SUPRARENAL GLANDS. EDWARD H. CAMPBELL, Arch. Otolaryng. **15**:517 (April) 1932.

Marine's definition of status lymphaticus is quoted as follows: "Status lymphaticus may be defined as a constitutional defect, usually congenital (though it may be acquired), dependent on an inadequacy of some function of the suprarenals, sex

glands and autonomic nervous system, associated with lowered resistance or increased susceptibility to a great variety of nonspecific, physical and chemical agents. Anatomically, it is characterized by delayed involution or hyperplasia of the thymus, hypertrophy and hyperplasia of the lymph glands and lymphoid tissue of the various organs, underdevelopment of the chromaffin, gonadal (suprarenal cortex, interstitial cells of the testes and ovaries) and cardiovascular systems and certain peculiarities of external configurations." There is also a tendency to leukocytosis, hypotension, longer coagulation time and a lowered blood sugar content and alkaline reserve. The leukocytosis shows a lowered polymorphonuclear with an increased lymphocytic count, giving a general lowered resistance to acute infections. Many patients dying of acute infections, such as endocarditis, meningitis, appendicitis and acute cholecystitis, show an enlarged thymus. Electric currents of low voltage more often kill patients with an enlarged thymus, and thymic enlargement has been found in a big proportion of persons who commit suicide, neurasthenic persons, gunmen and degenerate, epileptic and mentally defective persons. Deaths due to status lymphaticus have been known to be precipitated by slight infections or intoxications, various emotions, psychic shock, electric shock, shock from hypodermics or vaccination, traumatism, coitus, anesthesia, flogging, cold water, burns, bathing, muscular exertion, hemorrhage and drugs.

Status lymphaticus in Symmer's review of 5,652 autopsies was found in 8 per cent, being six times as common in females as in males. It is often familial.

Although there are cases of enlarged thymus without other involvement of the lymphatic constitution and conditions that indicate status lymphaticus without thymic enlargement, it is generally true that enlarged thymus is indicative of status lymphaticus. Investigators have gone so far as to question whether or not there is a normal size for a thymus, because of the great variation in size; hence too hard and fast rules of the association of thymic enlargement and status lymphaticus cannot be made. Moreover, the thymus changes size rapidly in some conditions; for instance, it diminishes rapidly in starvation, acute infections and pregnancy.

Enlargment of the thymus is most often diagnosed by means of the roentgen ray, but respiratory and cardiac changes so affect the configuration of the thymus shadow that real accuracy is problematic. Hasley and de Tomasi take thirty exposures on a roll of film in from ten to twenty seconds, thus giving pictures of the thymus in many of the respiratory and cardiac cycles. An enlightening study made by this method is in progress, which discredits much of the previous roentgenologic diagnostic work.

Williamson and Pearse have recently definitely shown that the thymus is composed of an aggregation of greatly plicated endothelial lymph tubules in the walls of which may or may not be lymph nodes. The thymus is in reality a part of the thyroid, and these lymph tubes are the blind terminals of the special thyroid lymphatics that have no connection with the other lymphatic system of the thyroid which carries the secretion containing the thyroxine and iodine directly into the blood channels. They further claim that this special secretory product of the thymothyroid system is responsible for the toxic state of exophthalmic goiter, and that it is the function of the thymus to detoxicate this subsance.

There is also a very close relationship between the thymus and the suprarenals. Marine, Manley and Baumann have expressed the belief that the thymic and lymphoid hyperplasias of childhood are manifestations of a functional underdevelopment of the suprarenal cortex and sex glands of varying intensity. It is also observed that enlargement of the thymus follows the removal of the suprarenals, and that since the hormones of the suprarenals act to protect the body in emergencies, such as flight, combat and infections, there seems to be a close relationship between enlarged thymus and the general findings incident to status lymphaticus. This is observed in man and experimental animals.

In autopsies in status lymphaticus some observers state there is always a hypoplasia of the suprarenals. Status lymphaticus is almost always found with pernicious anemia. Moreover, a lack of suprarenal secretions explains satisfactorily

the cause of sudden death in many of these cases. The enlarged thymus is only a symptom of status lymphaticus, which in turn is due to a deficiency of the secretions of the suprarenals.

Satisfactory tests to determine the presence of status lymphaticus must be developed, and experimental work with histamine is giving some promising results.

In view of the studies herein set forth, the logical treatment is suprarenal gland therapy, and though no satisfactory substance is on the market now, no doubt there will be one soon.

BARNETT, Chicago.

DIAGNOSTIC INTERPRETATION OF VARIOUS PHYSICAL SIGNS AND SYMPTOMS IN BABIES AT THE NEW YORK CITY HEALTH DEPARTMENT BABY HEALTH STATIONS. E. L. HERGERT, Arch. Pediat. **49**:471 (July) 1932.

The author attempts to interpret some of the usual symptoms and signs of disease in infancy and to connect them in their proper place in infant pathology.

WADDELL, University, Va.

CLINICAL MANIFESTATIONS OF HEAT AND EFFORT SENSITIVENESS AND COLD SENSITIVENESS (RELATIONSHIP TO HEAT PROSTRATION, EFFORT SYNDROME, ASTHMA, URTICARIA, DERMATOSES, NON-INFECTIOUS CORYZA, AND INFECTIONS). WILLIAM M. DUKE, J. Allergy **3**:257 (March) 1932.

A series of cases is presented in which the patients showed clinical reactions to heat and effort and cold. The reactions are manifested by the skin as urticaria and dermatoses; by the respiratory tract as asthma and bronchitis; by the heart as tachycardia and anginal pain, and by the stomach as nausea and vomiting, or there may be general symptoms such as syncope, heat prostration or shock. Physical allergy may be seen in persons who are sensitive to foods or pollens, with a history of sensitivity in the family, or in persons without an allergic history. In the latter, the condition is frequently subsequent to some acute illness and long convalescence which has unbalanced the heat-regulating mechanism. Other causes of sensitiveness to heat and cold may be fracture of the skull, hemorrhage of the brain, a stroke of lightning, cardiac or vascular disease which impairs the circulation in one or several parts of the body, disease in the suprarenals or the pituitary body, which alters the normal vascular tone, in the thyroid, which may alter the metabolic rate or in the intestine, which alters the absorption of histamine-like bodies, or disease which interferes with normal surface moisture or alters the caliber of the bronchioles.

The diagnosis is made with the aid of the history and objectively by exposing to the heat of a 1,500 watt nitrogen lamp, the skin of a normal area and an area distal to a tourniquet around the arm. In cold-sensitive patients ice is rubbed on the skin. A reaction occurs in from five to thirty seconds in a very sensitive person; when it is severe, relief can be given by using the agent opposite to that used in the test.

Treatment consists in avoiding conditions that will cause the illness or accustoming the patient to the conditions. Heat is applied to a person who is sensitive to heat until a reaction occurs; cold is then immediately applied. This is done as frequently as conditions warrant. In persons who are sensitive to cold, ice is rubbed on the skin until the reaction occurs, and then heat is applied. Tolerance for effort is developed through gradual subjection to the effect of effort. The ill effect of effort can be overcome frequently by the application of ice.

HOYER, Cincinnati.

HEAT SENSITIVENESS: REPORT OF A CASE TREATED SUCCESSFULLY. W. R. CAVEN, J. Allergy **3**:311 (March) 1932.

A patient, 31 years of age, was sensitive to exposure to warm water, a grate fire and hot and humid weather, and this sensitivity was noted after exercise.

The reaction was manifested by an edema of the hands, arms and legs, and in severe reactions, of the face. The symptoms had been present for fifteen years. The patient was treated unsuccessfully with typhoid vaccine, a thyroid preparation, anterior and posterior pituitary extract and hot baths. Success was attained by subjecting her to four diathermic treatments at intervals of two weeks. The current was turned on for one and one-half hours. She was then wrapped in blankets for two and one-half hours. At the first treatment the temperature was allowed to rise to 102.6 F. per rectum; at the subsequent treatments it was raised to 104.6 F. The patient passed through a hot, humid summer, danced and played tennis, with only slight signs of her former condition.

HOYER, Cincinnati.

A STUDY OF THE CALCIUM, PHOSPHORUS AND POTASSIUM OF THE SERUM AND SPINAL FLUID IN ASTHMA. D. M. LIERLE and R. A. SAGE, J. Allergy **3**:325 (May) 1932.

Specimens of the serum and spinal fluid of twenty patients with bronchial asthma were studied. The average values in the serum were: calcium, 10.65 mg.; phosphorus, 3.64 mg., and potassium, 19.0 mg. The calcium-potassium ratio of the serum was 1.79, and that of the calcium and phosphorus, 3.03. The average values in the spinal fluid were: calcium, 4.93 mg.; potassium, 10.74 mg., and phosphorus, 1.65 mg. The calcium-potassium ratio of the spinal fluid was 2.17; that for calcium and phosphorus, 3.26. The ratio between the calcium of the serum and that of the spinal fluid was 2.18; that for potassium, 1.78. In a group of seven patients having nonasthmatic conditions, the serum calcium was 10.7 mg. and potassium 20.26 mg.; the ratio was 1.84.

The technics used are described. HOYER, Cincinnati.

RECENT OBSERVATIONS IN THE STUDY OF ASTHMA DUE TO FOOD ALLERGY. A. V. STOESSER, J. Allergy **3**:332 (May) 1932.

A series of observations made in a girl, 12 years of age, who had had asthmatic attacks for eight years and who had had eczema previously are reported. She was sensitive to codfish, nuts, peas and eggs. The observations extended over a period of sixty-five days and were divided into twelve periods. Asthmatic attacks were produced by the giving of codfish. Diets high and low in carbohydrates, with and without the restriction of fluids, were used to study the response of asthma under these conditions. A ketogenic diet and a diet high in carbohydrates with a normal intake of fluid provided no protection against asthma. The reaction was more severe when the fluids were restricted with both the ketogenic diet and the diet high in carbohydrates. Full protection was obtained on a diet high in carbohydrates with a high intake of fluid when the patient was up and about indoors, but there was no protection while the child was out of doors and active. In the latter case the intravenous administration of a 25 per cent solution of dextrose controlled the allergic reactions as well as did the injection of epinephrine. Mild asthmatic attacks could be controlled by the oral administration of a 50 per cent solution of dextrose.

The author plans further work in this study. HOYER, Cincinnati.

EXOPHTHALMIC GOITER IN IDENTICAL TWIN GIRLS. FRANK C. NEFF, J. Pediat. **1**:239 (Aug.) 1932.

Cases of exophthalmic goiter in identical twin girls are reported, showing almost exactly the same physical and laboratory findings throughout. Several other cases are mentioned in which the same pathologic process became manifest in identical twins at the same time. LEMASTER, Evanston, Ill.

PRINCIPLES OF PHYSICAL AND HYDROTHERAPEUTIC TREATMENT OF ARTHRITIS. FRITZ MUNK, Brit. J. Phys. Med. **7**:60 (July) 1932.

The author discussed physical and hydrotherapeutic treatment as applied to various subacute and chronic conditions of the joints commonly grouped under rheumatic arthritis. He emphasized the necessity of early active and passive movement even before the pain has subsided. This is accomplished by the use of analgesics, the choice of which depends entirely on the degree of pain. The importance of the mental state of the arthritic patient was stressed. The method of treatment used was briefly outlined.

HARTMAN, Cleveland.

PHYSICAL METHODS IN PREVENTION OF JUVENILE RHEUMATISM. J. F. HALLS DALLY, Brit. J. Phys. Med. **7**:79 (Aug.) 1932.

From his experience and observations at the Saint Marylebone Children's Rheumatism Supervisory Centre, the author discusses diet, ultraviolet irradiation, hydrotherapy, sun and air baths, exercises and rest in the prevention of juvenile rheumatism.

HARTMAN, Cleveland.

LEUCOPLAKIA OF THE URINARY TRACT, WITH A REPORT OF A CASE. H. P. WINSBURY-WHITE, Brit. J. Surg. **20**:49 (July) 1932.

In the case reported there was a uniform change over the whole of the vesical mucosa except the trigone, which was ulcerated. This change consisted of the conversion of transitional epithelium into the squamous type. This condition is sometimes associated with squamous cell carcinoma. At present there are a number of theories as to the etiology of leukoplakia, the most acceptable of which is that the disease is due to a deficiency of vitamin A. Experimentally, a similar condition has been produced in rats, rabbits and guinea-pigs. The condition has also been found in an infant dying of a deficiency of vitamin A; there was widespread keratinization of epithelium in the glandular tissue and the mucous surfaces including the renal pelvis.

The epithelium of any part of the urinary bladder may be the seat of the leukoplakia. The gross appearance is that of single or multiple, raised, gray patches of varying sizes, or there is a uniform hyaline appearance of the mucous membrane. The condition occurs in infants as well as in adults. The symptoms are those of chronic infection, such as hematuria, frequency, terminal discomfort on micturition and turbidity of the urine. When leukoplakia occurs in the bladder, cystoscopy may prove unsatisfactory because of the fact that it is impossible to get the bladder to retain sufficient fluid for this examination to be carried out. Treatment at present is palliative, and consists of the use of radium and electrocoagulation.

SUMMERFELDT, Toronto, Canada.

THREE CASES OF OBSTRUCTION OF THE OESOPHAGUS. D. PATERSON, Proc. Roy. Soc. Med. **25**:711 (March) 1932.

The following three cases illustrate the principal nontraumatic forms of esophageal obstruction. The patients were admitted to the hospital about the same time.

A patient 8 days old was admitted on account of vomiting immediately after each feeding since birth. A small tube was passed through the mouth, but only reached down 4 inches (10 cm.) from the lips. A roentgenogram showed obstruction of the esophagus at the level of the third dorsal vertebra. A diagnosis of congenital atresia of the esophagus was made. The baby died shortly after admission.

A boy aged 4½ years had vomited all solid food since he was weaned. On examination, nothing abnormal was found except wasting. A roentgenogram showed a marked narrowing of the esophagus at the level of the seventh dorsal vertebra. A diagnosis of congenital narrowing of the esophagus was made.

A child aged 11½ years gave a history of vomiting every meal for the preceding six months. A roentgenogram showed dilation of the esophagus, with closure of the cardia. It was found that food remained in the esophagus for about five minutes before passing into the stomach. A diagnosis of achalasia of the cardia was made.

WILLIAMSON, New Orleans.

HAEMORRHAGIC HEPATITIS; AETIOLOGY UNKNOWN. F. J. POYNTON, W. W. PAYNE and REGINALD LIGHTWOOD, Proc. Roy. Soc. Med. **25**:785 (April) 1932.

This case was reported in a girl aged 5½ years. Two weeks before admission to the hospital she had a cold, and one week later she became jaundiced. On admission her condition resembled that in an ordinary case of catarrhal jaundice. The liver was enlarged and there were signs of pulmonary collapse of portions of the lower lobe of the right lung. Three days later she vomited and became suddenly worse, and then she became drowsy. One week from the day of admission she died in cholemia. All investigations made thirty-six hours before death and a full report of the autopsy are given in the report.

WILLIAMSON, New Orleans.

ENLARGEMENT OF PATELLA OF DOUBTFUL ORIGIN. H. A. T. FAIRBANK, Proc. Roy. Soc. Med. **25**:788 (April) 1932.

On examination the left patella of the patient, aged 12 years, was apparently enormously enlarged, being larger than the adult bone. The muscles were larger than those of the right leg. No disability was present. The roentgenogram showed that the patella itself was not so much enlarged, but above it and at both sides it was surrounded by separate fragments for a distance of three fourths of an inch (1.9 cm.). The condition called for no treatment.

WILLIAMSON, New Orleans.

OBESITY? NATURE. WILFRID SHELDON, Proc. Roy. Soc. Med. **25**:792 (April) 1932.

A boy of 11 years weighed 17 stone (97 Kg.) and was 64 inches (165.1 cm.) tall. The distribution of fat was diffuse, following no particular clinical pattern. The blood pressure was definitely raised (165 systolic and 95 diastolic), but otherwise there were no signs of organic disease. The boy had eight uncles, all of whom were policemen, so that bigness of build ran in the family, and the author felt that the child's size exemplified a family characteristic.

WILLIAMSON, New Orleans.

HIRSCHSPRUNG'S DISEASE, TREATED BY EXCISION OF THE INFERIOR MESENTERIC GANGLION. JOHN H. GIBBENS, Proc. Roy. Soc. Med. **25**:1225 (June) 1932.

This case was reported in a girl of 3½ years. Roentgen examination showed the typical signs of Hirschsprung's disease. The effects of physostigmine and acetylcholine were first tried, without any obvious clinical change. Then operation was advised, and the inferior mesenteric ganglion was excised. Nine months after operation, abdominal distention was much greater than previously, and peristalsis was marked.

WILLIAMSON, New Orleans.

PRIMARY HYPERTENSION IN A CHILD. ALAN MONCRIEFF, Proc. Roy. Soc. Med. **25**:1226 (June) 1932.

This case was reported in a child of 11 years, who entered the hospital on account of pains in the knees and a slight rise in temperature in the evening after which he complained of frontal headaches. The systolic blood pressure was then 145. The tonsils, which were large and unhealthy, were removed; this

relieved the headaches somewhat, but the blood pressure remained about 140 systolic and 100 diastolic. As no obvious cause could be found to account for the raised blood pressure this case appears to be one of primary or essential hypertension.

WILLIAMSON, New Orleans.

HYPOPHYSEAL DYSTOSIS. J. MOREAU, Arch. franco-belges de chir. **32**:697 (Sept.) 1931.

Moreau has gathered together thirty-four cases of the Schüller-Christian syndrome, and reports in detail a case observed by him. The reports collected from the literature are presented in extremely detailed abstracts, practically complete case histories. The early reports of Schüller and of Christian, together with the synthetic work of Rowland, are discussed fully, and many roentgenograms are reproduced from the original publications. The disease is characterized by four peculiarities: osteoporosis of the skull, exophthalmos, polyuria and pituitary dwarfism. The pathogenesis is probably a disturbance of the lipoid metabolism, characterized by hypercholesteremia and deposits of a xanthomatous character in the bones, particularly of the skull, and in the infundibulum, and less frequently in the skin and the internal organs.

The results of irradiation of the skull have been gratifying, and arrest of the condition or even recovery is reported. Spontaneous recovery occasionally occurs. Treatment by radiotherapy should be pursued diligently over a long period. In addition, a diet low in lipoids should be administered, and certain hypophyseal extracts are worth trying.

FREEMAN, Washington, D. C. [ARCH. NEUROL. & PSYCHIAT.]

RÔLE OF B. DIPHTHERIAE IN BRONCHOPNEUMONIA. H. BERNADON and P. LAMOTHE, Paris méd. **2**:43 (July 9) 1932.

There is no clinical or autopsy evidence that bronchopneumonia may cause an immediate predisposition to diphtheria, despite the frequency of these two diseases occurring in unison. The bacillus of diphtheria which is so often found in cultures from the nose and throat of patients suffering from bronchopneumonia is frequently only a saprophyte. Such patients, if they have had toxoid, are almost always immune. To sterilize the nose and buccal cavities the authors recommend solution 914 of Lereboullet and Gournay.

SANFORD, Chicago.

SEROLOGIC CHARACTERISTICS AND COLIPYURIA. MAX FRANK, Arch. f. Kinderh. **95**:217 (Feb. 5) 1932.

From a study of sixteen cases of colipyuria, the author makes the following observations. Two forms of colipyuria occur in childhood, one in which the patient's serum does not agglutinate, or agglutinates to only a slight degree, the organisms isolated from the urine, and one in which there is a high agglutination titer. The former is the most severe clinically. The difference may be due in part to the strain of the organism and in part to the patient's reactivity. The organism causing pyuria is always nonhemolyzing, so that this fact is no criterion as to the prognosis or the advisability of using vaccine. Therapy in these cases will be discussed in a further contribution.

LESLIE, Evanston, Ill.

PNEUMONIA IN CHILDHOOD: IV. PNEUMONIA AND APPENDICITIS. RUDOLPH KOCHMANN, Arch. f. Kinderh. **97**:157 (Sept. 9) 1932.

Symptoms resembling appendicitis, such as vomiting, constipation and abdominal pain, may occur at the onset of pneumonia in childhood, and may cause difficulty in diagnosis. Furthermore, actual appendicitis may arise during the course of pneumonia, as in two cases described by the author. Appendectomy is as fully indicated in this situation as at any other time.

LEMASTER, Evanston, Ill.

THE USE OF AMIDOPYRINE IN DIABETES INSIPIDUS. D. SCHERF, Wien. Arch. f. inn. Med. **22**:457 (July 15) 1932.

The use of large doses of a brand of amidopyrine caused a marked reduction of thirst and a pronounced diminution of water and sodium chloride diuresis in four of five cases of genuine diabetes insipidus.

The effect diminishes if the remedy is continued over long periods. It reappears, however, if the remedy is discontinued for a short period and then given again.

The body weight does not change much during the administration of amidopyrine, and the specific gravity of the urine increases only slightly. The effect of amidopyrine seems to depend on the sodium chloride content of the serum.

Phenobarbital does not alter the effect, and the diuresis-inhibiting action of pituitary is not interfered with.

The author recommends amidopyrine administered in intramuscular injections to combat the thirst of patients with diabetes insipidus.

The exact mode of action of the drug is not well known.

SCHLUTZ, Chicago.

Nervous Diseases

THE PEDIATRICIAN'S OBLIGATION: A PSYCHOANALYTICAL OBSERVATION DEALING WITH THE PREVENTION OF NEUROSES IN CHILDREN. BERTRAND S. FROHMAN, Arch. Pediat. **49**:339 (June) 1932.

The writer feels that the pediatrician should be stimulated to study the psychologic phase of his specialty. His function in the field of prevention should be enlarged to include the psychic as well as the physical.

WADDELL, University, Va.

DIAGNOSIS AND TREATMENT OF BEHAVIOR DISTURBANCES IN THE HOME. GRETE E. SEHAM, Arch. Pediat. **49**:354 (June) 1932.

Several cases are presented illustrating the diagnosis and therapeutic procedures that may be used in handling behavior disturbances in the home. The writer feels that the prevention and treatment of behavior disturbances divide themselves into two parts: first, the establishment of an adequate physiologic state and, second, the introduction of correct and specific psychologic stimuli. Diagnosis and treatment must be carried out in the home for two reasons: first, to discover all the factors which may cause undesirable behavior and, secondly, to introduce those which will affect it favorably. By way of illustration, some of the factors which influence eating and sleeping are cited.

WADDELL, University, Va.

HEMIPLEGIA. SYDNEY NUSSBAUM, Arch. Pediat. **49**:392 (June) 1932.

Infantile cerebral palsies are best classified as prenatal, natal and acute acquired, according to the period of life during which the lesion occurs.

Three cases of hemiplegia belonging to the acute acquired palsies are reported. The first case was due to hereditary syphilis with cerebrospinal involvement in the meningovascular stage. The second case occurred as a complication of pertussis. The third case presented hemiplegia and epilepsy as permanent sequelae of epidemic encephalitis.

General clinical and pathologic features are discussed, and individual characteristics of the three conditions in which the hemiplegias occurred are outlined.

WADDELL, University, Va. [AUTHOR'S SUMMARY.]

ALTERED POSITION OF CHILDREN AS A FACTOR IN BEHAVIOR, WITH PARTICULAR CONSIDERATION OF NEGRO CHILDREN. IRA S. WILE, Arch. Pediat. **49**:494 (Aug.) 1932.

The changed position of the child in the family as compared to the older order of things is discussed, particularly as these changes affect the Negro child. The writer believes that many of these changes are responsible for behavior problems so frequently seen in Negro children. The nature of such social changes are discussed in detail. The writer feels that there is certainly increased need for social action in the development of the status of children in colored families. Society as a whole must recognize the relationship of health, education, recreation and occupation as factors in promoting the welfare of colored children.

He feels that colored boys and girls cannot be condemned for delinqency until society is nondelinquent in fulfiling its responsibilities to the growing generation.

WADDELL, University, Va.

RECENT EPIDEMIC OF POLIOMYELITIS: NEW YORK CITY'S EXPERIENCE. GIUSEPPE PREVITALI, Arch. Pediat. **49**:540 (Aug.) 1932.

The efforts of New York City in 1931 to cope with the epidemic of poliomyelitis are described in detail. The writer discusses steps that were taken to further prompt diagnosis, hospitalization, better treatment, forced rest and follow-up and after-care. He feels that New York was much better prepared to cope with the epidemic in 1931 than on previous occasions.

Figures from the department of health giving results in ninety-six patients treated with convalescent serum and in sixty-one with antipoliomyelitic serum are presented. Control cases are also reported. As far as can be judged by statistics of the New York Department of Health, it appears that when such studies are made with controls, there is very little difference, in the course of the disease and in the after-effect, between the patients who receive treatment and the controls, even in the preparalytic stage.

WADDELL, University, Va.

REFLEXES AND OTHER MOTOR ACTIVITIES IN NEW-BORN INFANTS: A REPORT OF ONE HUNDRED AND TWENTY-FIVE CASES AS A PRELIMINARY STUDY OF INFANT BEHAVIOR. L. BEVERLY CHANEY and MYRTLE B. MCGRAW, Bull. Neurol. Inst., New York **2**:1 (March) 1932.

The authors have carefully studied the reflex reactions in one hundred and twenty-five apparently normal infants, examining reflexes at birth and on each succeeding day for ten days. Their observations are too lengthy for summary, except to point out that individual differences are present in the simplest type of reflex. Total absence of any active reflex is strongly suggestive of a pathologic condition.

BEVERLY, Chicago.

BLOOD SUPPLY OF THE GLIOMAS: ITS RELATION TO THE TUMOR GROWTH AND ITS SURGICAL SIGNIFICANCE. CHARLES A. ELSBERG and CLARENCE C. HARE, Bull. Neurol. Inst., New York **2**:210 (July) 1932.

There is a close connection between the distribution of blood vessels and the manner in which gliomas infiltrate the tissue. In the astrocytomas and the medulloblastomas, the largest number of vessels occur in the central part of the growths. There is no increase in the number of arteries in the adjacent white matter. In glioblastoma multiforme the peripheral areas of the neoplasm contain the largest number of arteries. The differences in the blood supply and the manner in which the brain is involved in the astrocytoma and the medulloblastoma, on the one hand, and in the glioblastoma multiforme, on the other, are expressions of the varying degree of malignancy.

BEVERLY, Chicago.

VENOUS AND ARTERIO-VENOUS ANGIOMAS OF THE BRAIN: A CLINICAL AND ROENTGENOGRAPHIC STUDY OF EIGHT CASES. SAMUEL BROCK and CORNELIUS G. DYKE, Bull. Neurol. Inst., New York **2**:247 (July) 1932.

Eight cases of intracranial angioma are described with a general discussion of vascular lesions of the brain.

There are two types of vascular lesions found—hemangioblastomas and vascular malformations. The latter are divided into telangiectases, venous angiomas and arteriovenous angiomas.

In four of the cases there were extracranial lesions. The important ocular symptoms were homonomous hemianopia and unilateral exophthalmos. In the arteriovenous variety, cardiovascular phenomena were present, including enlarged carotids, a systolic mitral murmur and mild tachycardia. Roentgenograms demonstrated intracerebral calcification and enlarged, tortuous vascular grooves in the bones of the skull.

Treatment consisted in ligation of the vessel, roentgen treatment and removal of the cerebral lesion.

BEVERLY, Chicago.

DEMONSTRATION OF NORMAL CEREBRAL STRUCTURES BY MEANS OF ENCEPHALOGRAPHY. CORNELIUS G. DYKE and LEO M. DAVIDOFF, Bull. Neurol. Inst., New York **2**:331 (July) 1932.

A careful study of cerebral structure by means of encephalography in fifty cases led the authors to conclude that one sometimes finds apparent defects in bodies of the lateral ventricle and the roof of the fourth ventricle which are caused by the choroid plexus. The presence of these defects in the ventricles depends on the size of the choroid plexus and does not appear to be associated with any particular disease.

BEVERLY, Chicago.

PSYCHONEUROSES IN CHILDREN. HERBERT B. WILCOX, Bull. New York Acad. Med. **8**:502 (Aug.) 1932.

Among 5,500 admissions to the Babies Hospital, there were 55 instances (1 per cent) in which there were behavior problems and neuroses of some sort. Analysis of 1,100 cases in private practice showed 96 children (9 per cent) with neuroses and behavior problems as the major complaint.

In the whole group, 60 per cent were boys, 5 per cent more in the hospital than in private practice. Age was not of much importance in the series reported. Sixty-nine per cent of the entire group showed no nutritional anomalies. There were 167 instances of bad parental management, the frequency being 3 times greater in the private than in the hospital group. There were 50 instances of neurotic parents, these instances being 4 times as frequent for private patients as for hospital patients. Overcontrol occurred in 23 cases; that is, it was 7 times as frequent in private as in hospital patients. Neglect occurred in 26 cases, 5 times as frequently in private as in hospital patients. Shock occurred in 13 cases, the distribution being equal in hospital and in private groups.

Behavior problems are complicated because the child is not a single unit. The child's rest must be taken into account, and its nurse, parent, grandparent, maiden aunt or elder brother. The problem in the child is a reflection of adult errors in administration. As to the relative responsibility of pediatrician and psychiatrist: 1. The pediatrist must be psychiatrically minded in order to break up the vicious circle in the child-parent scheme. 2. Children, even in infancy, show evidences of neuroses. These may be transitory or cause permanent damage, but all these children need careful guidance through parental understanding. 3. Child behavior problems will not occur if the parents are educated to discipline and control the child. "The pediatrist, being first on the ground, has the opportunity to foresee, forewarn and so perhaps prevent the need of psychiatric advice. If unable to avoid the necessity he can at least see that assistance is early sought."

A comprehensive discussion of the genesis of behavior problems in the child is followed by the explanation of the neuroses in New York children, which applies

equally well to other communities in American life: "Overstimulation by noise, competition, parental anxieties, apprehension, or ambition; by standardization of school and exercise; by complicated amusements—in short, by the lack of that thing most necessary to unmyelinized youth—quiet."

JAHR, Omaha.

CERTAIN PEDIATRIC DOUBTS ABOUT MODERN PSYCHIATRY. BRONSON CROTHERS, Bull. New York Acad. Med. **8**:515 (Aug.) 1932.

The aims and results of the specialized (behavior) clinic are better understood by teachers, social workers and parents than by doctors in general. The pediatrician has had no training in formal psychiatry and therefore pays little attention to those who urge him to acquire it. At present, however, the pediatrician is forced to the appreciation of team work. Unless his interest in this is developed, the pediatrician will be looked on as an advisor on the physical status of the child, while information on the mental and emotional development will be sought from other groups. This will mean chaos.

The medical profession should realize that psychiatrists, as they now practice, are interested in people, especially children who are neither feebleminded nor insane, but who are making less of life than they should. On the other hand, it is impossible to know what psychiatrists, as a group, want to do. They must be prepared to control propaganda or disown it. Present psychiatric information is too dilute and handed out to doctors, parents, teachers and social workers in the same form. A more definite medicopsychiatric literature is badly needed to satisfy the comprehension of the physician. This is the duty of psychiatry. If the psychiatrist does not offer a crystalized program, the pediatrician must find the way to utilize the information elaborated by the psychiatrist, psychologist and teacher. Indifference to the subject is a most pernicious attitude in the light of the potential benefits which are included in the knowledge now available.

JAHR, Omaha.

ACTIVE EXTERNAL HYDROCEPHALUS WITH REPORT OF THREE CASES. W. J. GARDNER, J. Nerv. & Ment. Dis. **75**:601 (June) 1932.

Three cases of external hydrocephalus of unknown etiology were diagnosed with the aid of the encephalogram. Recovery occurred after subtemporal decompression.

JAHR, Omaha.

POLIOMYELITIS. J. J. SANDS, J. Nerv. & Ment. Dis. **75**:601 (June) 1932.

Poliomyelitis is an acute infectious disease marked by a pathologic condition of the nervous system dominantly in the anterior horns of the spinal cord, with severe meningitis chromatolysis, neuronophagia and vacuole degeneration of the cells of the anterior horn and of the nerve centers in the medulla.

The initial signs of the disease are marked meningeal and cerebral irritation with fever and prostration.

The treatment which is at present considered most effective is (1) the injection [intravenously and intramuscularly] of convalescent serum as soon as possible after the diagnosis is made, (2) the alleviation of edema of the brain by injection of hypertonic dextrose solution, (3) spinal drainage, (4) the combating of the symptoms and (5) the protection of paralyzed muscle by appropriate support.

There should be prophylactic segregation and immune serum or whole blood should be injected intramuscularly every two or four weeks during the period of epidemic.

JAHR, Omaha.

POSTENCEPHALITIC ORDINARY AND EXTRAORDINARY CHILDREN. EARL D. BOND, J. Pediat. **1**:310 (Sept.) 1932.

The author discusses the work done at the Franklin School of the Pennsylvania Hospital on behavior problems caused by encephalitis, cerebral injury and emotional

trauma in children. Good results have been obtained by the attention of understanding teachers and by long interviews in which the child tells an uncritical adult what is on his mind. The pediatrician is urged to apply these principles of behavior problems in their incipiency to the "spoiled" child as well as to children in the postencephalitic state.

LEMASTER, Evanston, Ill.

POLIOMYELITIS IN THE PREPARALYTIC STAGE WITH A DISCUSSION OF TREATMENT. O. N. TORIAN and MATTHEW WINTERS, J. Pediat. **1**:326 (Sept.) 1932.

The clinical aspects and treatment of the preparalytic stage of poliomyelitis are discussed. Attention is called to the importance of the "spine sign" in all febrile cases in which the diagnosis is not clearly evident. Because of occasional sporadic cases, doctors must constantly think of the preparalytic stage of poliomyelitis, even in the absence of an epidemic. Cases are cited to show the value of the "spine sign" and of lumbar puncture in diagnosis and differential diagnosis.

LEMASTER, Evanston, Ill.

EARLY ACUTE ANTERIOR POLIOMYELITIS WITHOUT AN INCREASE OF CELLS IN THE SPINAL FLUID. SIDNEY O. LEVINSON, J. Pediat. **1**:337 (Sept.) 1932.

The author reports five cases of preparalytic poliomyelitis in which the clinical diagnosis was confirmed by muscular weakness or paralysis, but in which there was no increase of cells in the spinal fluid. Similar findings reported by other observers are discussed. This condition is explained by the pathologic evidence that inflammation of the cord precedes meningeal inflammation. Hence cases of preparalytic poliomyelitis may be seen with clinical manifestations of involvement of the cord, but without the production of an increase of cells in the spinal fluid.

LEMASTER, Evanston, Ill.

BLOOD GROUPING IN POLIOMYELITIS. E. B. SHAW, H. E. THELANDER and K. KILGARIFF, J. Pediat. **1**:346 (Sept.) 1932.

A determination of blood groups in one hundred cases of poliomyelitis in San Francisco is presented, together with a review of a similar study of seventy-eight cases from the lower Rhine district. Several normal groups are included for comparison. In none of these series were any representatives of group I encountered. The representation of the other blood groups was within fairly normal limits.

LEMASTER, Evanston, Ill.

THE POLIOMYELITIS EPIDEMIC. HERMAN T. PECK, New York State J. Med. **32**:854 (July 15) 1932.

Of 4,080 cases of poliomyelitis reported in New York City during the first ten months of 1931, only 34 occurred previous to July 1. Early in the epidemic the commissioner of health called a meeting of the medical advisory board of the health department, with representatives from the 5 county medical societies. The publicity regarding the disease was so conducted that the fear and apprehension of the epidemic of 1916 were largely avoided.

Of the patients, 3,112, or 76 per cent, received hospital care; 466 died, making a mortality of 11 per cent as compared to that of 27 per cent in 1916. Fifty-three per cent of the patients were under 5 years of age.

Of 4,080 cases, only 95 families had 2 cases, and 3 families had 3 cases. "Only 6 cases could be considered purely contact cases according to our present conception of secondary cases as applied to other contagious diseases."

AIKMAN, Rochester, N. Y.

ACUTE ANTERIOR POLIOMYELITIS. BENJAMIN KRAMER, New York State J. Med. **32**:855 (July 15) 1932.

Ninety-eight patients were admitted to the service for infantile paralysis of the Jewish Hospital of Brooklyn during the epidemic of 1931. Special study was made of the recognition of the disease in the preparalytic stage, of the value of tested convalescent serum and of the evaluation of the Drinker respirator.

Ninety per cent of the patients were under 10 years of age. Fifty-four were seen in the preparalytic stage. Of the latter, twenty-four received no serum, and paralysis developed in only six. The other thirty received serum, and some form of paralysis developed in twelve.

Of forty-four patients admitted in the paralytic stage, eight showed bulbar, twelve bulbar spinal and twenty-four purely spinal involvement. The respirator was of value only in the spinal type of respiratory paralysis and not in the bulbospinal or bulbar type.

Of ten patients treated in the respirator, eight died. Two patients who survived had paralysis of the spinal type. The extent of residual paralysis is not stated.

AIKMAN, Rochester, N. Y.

THE CHVOSTEK SIGN IN INFANCY: ITS BEARING ON MENTAL CHARACTERISTICS. M. SCHACHTER, Arch. de méd. d. enf. **35**:346 (June) 1932.

There has been an opinion prevailing for many years, particularly among German pediatricians, that the future of infants showing the so-called facial sign is likely to be discouraging, with pronounced lack of neuromental development. Following the studies in this unusual field made by Thiemich and supported by Finkelstein, Nobécourt asserted that, sooner or later, children exhibiting over long periods the classic Chvostek reaction may be expected to present evidences of deterioration and become neuropathic or epileptic. Thiemich declared that in probably two thirds of the cases some such disaster may follow; in this he was sustained by Heubner.

The question of spasmophilia does not enter the discussion; the psychologic and pedagogic aspects alone of those subjects showing the facial sign are considered. Schachter maintains that the conclusions cited were based on too little material and with too much confidence on histories obtained from parents, teachers and guardians with respect to the mental integrity of these children.

Of 4,943 children examined by Naglo, 1,041, or 20 per cent, had a positive Chvostek sign, with the sexes about equally divided. The children were registered in the outpatient clinic and were suffering from various diseases. Normal children were found to reveal this sign in 4 per cent of subjects, it being rare in the very young and increasing in frequency so that between the ages of 8 and 13 fully one third may respond to this test.

Up to the age of 2 or 3 years the sign may be accepted as an evidence of tetany, but, the writer believes, under no circumstances can a Chvostek reaction be interpreted among older children as an evidence of inferiority. This view is largely based on the extensive investigation of Hummel and Jung at the University of Jena from which the author makes extensive citations.

AMESSE, Denver.

PROTRACTED TYPE OF MENINGOCOCCUS MENINGITIS. C. COCCHI, Riv. di clin. pediat. **30**:481 (April) 1932.

The author reports the case of a child 3½ years old who had meningococcic meningitis with symptoms and spinal fluid findings lasting over a period of four months. The disease occurred in exacerbations and is described as pseudomalarial. The first attack was mistaken for rheumatic fever and was treated with salicylates. The fever disappeared, but recurred three days later. A lumbar puncture established the diagnosis. Treatment was with serum and by autogenous vaccine.

HIGGINS, Boston.

Spastic Paraplegia in the Lower Extremities in a Child Six Years of Age. Sara Satanowsky, Arch. argent. de pediat. **3**:293 (May) 1932.

The condition occurred at birth in connection with a difficult extraction, and was due to an injury at birth. Flaccid paralysis of the lower extremities developed immediately after birth, and there was loss of all the sphincters. The flaccid paralysis gradually changed to a spastic paralysis. There were no particularly marked sensory changes in the lower extremities, but there were considerable trophic disturbance of the musculature and complete loss of sphincter action. The neurologic lesion at the time of the injury was probably in the sacrolumbar plexus.

Schlutz, Chicago.

Acute Sydenham's Chorea and Delirious State. J. M. Estape and B. Delgado Correa, Arch. de pediat. d. Uruguay **3**:69 (Feb.) 1932.

The condition reported occurred in a child of 8 years with marked contitutional and emotional background. The generalized choreic movements apparently began with an emotional shock and were complicated by a persistent delirious state expressed particularly in hallucinations and various manifestations of fear. There were no rheumatic manifestations or cardiac complications. There were no evidences of poliomyelitis in the case and no history of any other infectious or contagious disease.

The author believes that the case would classify under a type of condition described by Dupré as psychopathic, hyperemotional constitution. The author also believes that the case could possibly be explained on the theory expressed by Cannon, that in these emotional shocks a hyperadrenalemia occurs, which leads to peculiar biochemical changes which in turn influence the endocrine and the sympathetic system.

Schlutz, Chicago.

Spontaneous Meningeal Hemorrhage—Recurring Form. Julio A. Bauza, Arch. de pediat. d. Uruguay **3**:320 (July) 1932.

This condition occurred in a girl 11 years old and was a recurrence of a similar disorder which had appeared five years previously. The onset was sudden and was accompanied by epileptiform convulsions of the jacksonian type. The entire left side of the body was involved. There were intense rigidity of the neck, a positive Kernig sign, exaggerated reflexes and a positive Babinski sign. Lumbar puncture showed hemorrhagic fluid. The girl had scanty and painful menstruation but was otherwise in very good health. There was no cardiac or renal disease.

The author believes that the condition was due to rupture of a small vein in the pia mater, with resulting extravasation into the subarrachnoid space in the rolandic zone.

The patient made a complete recovery. The causative factor is very obscure, but is believed by the author to lie in a constitutional alteration of the blood vessels leading to increased fragility and permeability. Recovery from the condition is very slow.

Schlutz, Chicago.

Two Cases of Amaurotic Familial Idiocy. Santiago Cavengt, Pediatria españ. **20**:33 (Feb.) 1931.

The author reports two cases of amaurotic familial idiocy in two members of the same family. Of four children, the first and third, both girls, were normal, while the second and third, both boys, presented the classic symptoms of Tay-Sachs' disease.

Careful investigation showed that the family was not Jewish in origin and that the medical history was essentially unimportant. The ocular changes in these two patients deviated somewhat from the classic picture of Tay-Sachs' disease in that the characteristic cherry-red spot in the macula was absent.

However, there were definite atrophic and degenerative changes in the macula and optic nerve resulting in blindness.

The author presents a detailed discussion of the pathology of Tay-Sachs' disease.

SCHLUTZ, Chicago.

IS THE PSEUDOTETANUS OF ESCHERICH AN ENCEPHALITIC CONDITION? MARIA NIERMAN, Arch. f. Kinderh. **96**:108 (April 8) 1932.

The author presents the case of a girl, aged 4 years, in whom, following a short period of diarrhea and fever, there developed change in facial expression, difficulty in taking food, increasing stiffness of the legs and attacks of cyanosis with threatening suffocation. Examination showed the head turned to the right, stiffness of the neck, ptosis of the right eyelid, typical risus sardonicus with inability to open the mouth, exaggerated patellar reflexes and a positive Babinski sign on the right. When the patient was left alone she showed some interest in her surroundings and played a little, but when disturbed, she stiffened out, showing opisthotonos, cyanosis and dyspnea. Following lumbar puncture, which showed increased pressure and a blood sugar of 118 mg. per hundred cubic centimeters, all symptoms were exaggerated, and the patient lay "stiff as a stick" for six days. At this time a serum reaction from the tetanus antitoxin developed, and at the same time the child began to improve. After three weeks she had entirely recovered. This picture corresponds closely with that described by Escherich as pseudotetanus. After two years, however, the child returned with chorea; this raises the question of whether the chorea was of the Sydenham type or the sequel that of encephalitis. To test the latter theory, fluid obtained by lumbar puncture was injected into the cornea of a rabbit. The result was interpreted as a positive reaction indicating the presence of the encephalitis virus. The child recovered from the chorea following treatment with phenyl-ethyl-hydantoin, and there has been no recurrence in two years.

LESLIE, Evanston, Ill.

THE PATHOLOGY OF BRAIN ABSCESSES IN INFANCY. KURT SAMSON, Arch. f. Kinderh. **96**:215 (June 3) 1932.

Three cases of brain abscesses in infancy are reported, together with a review of three cases from the literature. Pneumonia appeared to be the causative factor in half the patients and a suppurative cutaneous infection in the other half. The latent period lasted from two to three months before the sudden onset of the manifest stage, characterized by variable cerebral symptoms and enlargement of the head. Lumbar punctures yielded sterile fluid containing a normal amount of sugar, increased protein with the globulin proportionately greater than the albumin and an increased leukocyte count. Emphasis is placed on the importance of encephalograms showing widening and distortion of the lateral ventricles and of ventricular punctures in which the fluid is yellow, coagulates spontaneously and contains a normal amount of sugar, a high cell count and an increased amount of protein, mostly globulin. After these studies, further punctures of the brain are indicated in order to locate the abscess before the performance of surgical drainage. Staphylococci were found in all abscesses. None of the patients survived.

LEMASTER, Evanston, Ill.

THE CLINICAL PICTURE AND THERAPY OF POLIOMYELITIS AMONG ADULTS. F. W. BREMER, Deutsches Arch. f. klin. Med. **173**:58 (April 20) 1932.

The author concludes, from his experience with fourteen cases of poliomyelitis in adults, that the disease in adults differs widely from that seen in children. The preparalytic stage is longer. The paralysis is not sudden in onset but ensues step by step, often over a period of weeks. Symptoms of meningeal irritation are more pronounced than in children. "Abortive" cases are more frequent. In contrast to its action in childhood, immune serum (from Pettit of the Pasteur Institute of Paris) was effective after the onset of paralysis.

DOOLEY, Kent, Conn.

SENSITIVITY TO EPINEPHRINE IN DISEASE OF THE BRAIN. S. MOLNAR and A. LESZLER, Deutsches Arch. f. klin. Med. **173**:109 (April 20) 1932.

In a study of blood pressure in thirteen cases of tumor of the brain, the authors found a disturbance of sensitivity to epinephrine which they attribute to direct or indirect lesion of the sympathetic center.

DOOLEY, Kent, Conn.

SPINAL FLUID SUGAR IN CHILDHOOD. R. MATOSSI, Jahrb. f. Kinderh. **135**: 175 (April) 1932.

Over 500 specimens of spinal fluid were examined. The amount of dextrose varied between 40 and 70 mg. per hundred cubic centimeters. Administration of sugar by mouth caused only slight elevation; parenteral administration caused a considerable rise. If the cell content is low, there is little change in sugar content either in the refrigerator or in the incubator. In fluids rich in cells, there is marked decrease. Marked elevation in sugar was observed in intoxication, uremia and increased intracranial pressure. Moderate elevation was observed in encephalitis, poliomyelitis and infectious diseases with meningismus. Low values of sugar were observed in all fluids rich in cells, such as those in tuberculous or purulent meningitis. However, in the early stages of these diseases, the sugar content may be high.

CLAUSEN, Rochester, N. Y.

POLIOMYELITIS. D. HERDERSCHEE, Nederl. tijdschr. v. geneesk. **76**:1185 (March 5) 1932.

In 1929 there occurred in Amsterdam forty-one cases of poliomyelitis, of which twenty-four came from the western part of the city and seventeen from the eastern part; in 1930 these figures were thirty-eight, thirteen and twenty-five, respectively. The last widespread epidemic of the disease in Holland was that of 1912. The probable cause of the periodical appearance of diseases like poliomyelitis and the many difficulties in the diagnosis are discussed. The question of the treatment with convalescent serum in the preparalytic stage is extensively discussed. The author himself, though he had had extensive experience in the hospital, had never seen a case of poliomyelitis which was absolutely in the preparalytic stage. The author gives some indications as to how the disease in this stage may be recognized, so that serum treatment may be begun as soon as possible.

VAN CREVELD, Amsterdam, Holland.

NONPURULENT ENCEPHALITIS IN SMALL CHILDREN. W. J. C. VERHAART, Nederl. tijdschr. v. geneesk. **76**:3262 (July 2) 1932.

The author described two cases of probable toxic degenerative encephalopathy observed in young Chinese infants. The anatomic findings were: extensive demyelinization in the white matter of the brain, with hypertrophy and hyperplasia of the macroglia and formation of numerous fat granules of the same localization. In the cerebral cortex there was degeneration of the nerve cells, with macroglic reaction. Subependymal cell-reaction, perivascular calcifications and concrements were also found. These changes resemble those found in Schilder's and in Virchow's diseases.

VAN CREVELD, Amsterdam, Holland.

Diseases of the Ear, Nose and Throat

DIAGNOSIS AND TREATMENT OF SINUSITIS. FREDERICK E. HASTY, South. M. J. **25**:1202 (Dec.) 1932.

The author concludes that proper care of infants and children will do much to prevent sinusitis in later life. Care of sinusitis in children consists mainly of general management and nonoperative treatment. Allergy plays an important part in sinusitis in patients of all ages. All possible information regarding the type of

pathologic process in a particular case should be at hand before any treatment or surgical intervention is advised. The majority of diseased antrums can be cured by the injection of iodized oil. So-called "piddling" operations usually do little if any good.

SCHULTZ, Chicago.

ADENOIDS AND ADENOTOMY IN FIRST WEEKS AND MONTHS OF LIFE. H. NEUMANN, Monatschr. f. Ohrenh. **66**:922 (Aug.) 1932.

Neumann says that adenoid growths in infants who feed at the breast or on a bottle may obstruct nasal breathing to such a degree that sucking is interfered with and nutrition is disturbed. In the majority of these cases medical treatment will permit postponing adenotomy till the end of the first half year or year or at least beyond the first weeks or months of life, but in some cases surgical intervention becomes an immediate necessity. The author thinks that adenotomy is indicated if a palpable tumor filling the nasopharynx has been ascertained, if interference with feeding and nutrition results from the obstruction of nasal breathing, or if there is retarded development attributed to adenoids by the attending pediatrician. Three distinct types of tonsils occur in nurslings. The first is simple hyperplasia without accumulation of secretion. The second is irregular hypertrophy showing the beginning of the villous form frequently seen in older persons. This form usually has a heavy tenacious mucoid secretion and is often accompanied by nasal hypersecretion. The third form has a relatively small tumor, but heavy secretion in the nose and nasopharynx. Diagnosis in infants only a few weeks or months old is difficult. The author employs palpation with a sound introduced by mouth and indirect palpation with the finger to determine the resistance when the uvula is pressed back. The pressing down of the rear part of the tongue with a small laryngeal mirror frequently permits seeing the adenoid growths in the initial moment of the vomiting reflex. The author recommends instillation of oil for one or two weeks before adenotomy whenever possible. He describes the technic of performing the operation with his modification of Beckman's curet. This curet possesses a prong which is pushed into the tumor, holding it firm. It is essential to use a sharp instrument and perform the operation in one stroke. An anesthetic is not used in children under 1 year of age. Hemorrhage is slight. The results of this intervention in nurslings are usually excellent.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

CONGENITAL MALDEVELOPMENT OF THE LARYNX. ANNELISE ROHRMANN, Virchows Arch. f. path. Anat. **283**:304, 1932.

The anomaly occurred in a new-born girl with multiple malformations, and consisted in rudimentary development of the epiglottis; in place of the latter there was a group of cartilaginous nodules.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

TEETH IN THE NOSE. H. LEICHER, Ztschr. f. Laryng., Rhin. (Teil 1: Folia otolaryng.) **22**:445 (June) 1932.

Leicher reports four cases in which teeth were found in the nose. In most cases, these rare manifestations are due to inversion of a tooth, so that in place of the inverted tooth that has grown into the nose, there is a gap in the teeth. The first case was one of inversion of the tooth pulp. The anamnesis indicated that it was caused by trauma at the age of 3½ years, but the stage of development of the permanent incisors at this age makes a turning of 150 degrees as a result of trauma improbable. Further, a sister of the patient also had a gap in the permanent teeth without a history of trauma. Roentgenography in her case indicated that the missing incisor was inverted in the hard palate. This was probably a case of congenital anomaly of development. The second case concerns a woman with a supernumerary tooth. The third case concerns a boy, aged 8, with a cleft palate. Operation on the cleft palate disclosed the root of a tooth which extended

about 4 mm. into the nose. In the fourth case, the tooth did not grow into the nose, but merely lodged there. Presumably it was pushed into the maxillary sinus during attempted extraction, where it caused suppuration. In the course of irrigation of the sinus, the tooth arrived in the nose through a large opening in the lateral nasal wall.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

TONSILLECTOMY IN INTERNAL DISEASES AND THE PROBLEM OF ORAL SEPSIS. L. POLEDNAK and J. HORAK, Sborn. lék. **36**:19 (July 11) 1930.

The author reviews the literature in detail and cites tables and classifications of diseases directly and indirectly associated with oral sepsis. In his series 114 patients were watched for a period of eight years. The effect of tonsillectomy and dental extraction was checked in rheumatism, heart disease, nephritis, septicemia, nervous diseases, bronchial asthma, disease of the thyroid gland and anemia. Operation was carried out only in those cases in which both the internal and local indications warranted operation. The oral treatment was usually radical, that is, tonsillectomy and extraction. All local symptoms disappeared; generally the best results were obtained in acute rheumatic conditions of the joints, rheumatic disease of the heart, oral sepsis and secondary nephritis.

STULIK, Chicago.

Diseases of the Skin

ACNE VULGARIS, HEREDITARY IN THE ETIOLOGIC BACKGROUND. JOHN H. STOKES and ALLEN D. KING, Arch. Dermat. & Syph. **26**:456 (Sept.) 1932.

A study of the histories of 143 patients with acne vulgaris and of 100 normal physicians and nurses was made to find evidence of a familial incidence of this disease. The authors found definite evidence that a familial predisposition exists in this disease. This is an important consideration in the treatment and prognosis. They also discuss the various theories concerning the complete etiology. This familial factor does not seem to follow a simple mendelian distribution.

EKERMEYER, Iowa City.

RINGWORM OF THE SCALP: TREATMENT WITH THYMOL AND OIL OF CINNAMON. ELMER C. LOOMIS, Arch. Dermat. & Syph. **26**:495 (Sept.) 1932.

The author used a mixture of 0.5 per cent thymol and 1 per cent oil of cinnamon in the treatment of ten patients with clinical ringworm of the scalp. Eight patients showed no improvement, and two were considered cured. Loomis believes that the treatment described is of little value in the treatment of this disease, and that the only satisfactory management is the method by which the infected hairs are removed.

EKERMEYER, Iowa City.

THE INFANTILE ECZEMA PROBLEM. LEWIS WEBB HILL, New England J. Med. **207**:655 (Oct. 13) 1932.

The author discusses the various theories advanced as to the causation of infantile eczema, including those of high fat, split proteins, milk per se, sensitization of the nursing infant to foods eaten by its mother and external sources of irritation. The author reports that good results were obtained in some severe cases by the stopping of all milk and the feeding of a soy bean preparation, but he is willing to admit that there is more to the problem than "omitting milk from the diet," "scratching the skin" or "local treatment."

GENGENBACH, Denver.

INFANTILE HIRSUTISM TREATED BY ROENTGENOTHERAPY. MAHAR and FALCOZ, Bull. et mém. Soc. de radiol. méd. de France **18**:314 (July) 1930.

Mahar and Falcoz present a case of infantile hirsutism due to hyperplasia of the suprarenal glands following mumps. The condition was treated by roentgen

therapy, with improvement. Three years ago at the age of 7 the child contracted mumps. This was of a rather benign course. Shortly after the healing of the mumps there was a rapid increase in the weight of the patient, and the entire body became covered with coarse, stiff, black hairs, more marked on the back, extremities, upper lip and chin. During the same time the child showed signs of advanced maturity. In 1929 mixed organotherapy (thyroid, pituitary) was instituted, but without much result. At the age of 10, the patient had an abundant growth of black hair distributed over practically the entire body with a mustache and beard. There was no pathologic pigmentation, and on palpation there was no evidence of an abdominal or lumbar tumor. The basal metabolism was minus 16. Roentgenograms of the skeleton failed to reveal any pathologic changes. A diagnosis of hirsutism was made (a term first used by Apert for the designation of a syndrome of obesity and hypertrichosis). It was assumed that the condition was the result of inflammatory changes of the suprarenal glands following mumps. The application of roentgen rays with medium voltage (22 cm. spark gap, with 0.2 mm. copper and 1 mm. aluminum filters) over both suprarenal glands led to remarkable improvement. The treatment was started on Feb. 24, 1930, the dose amounting to 300 roentgens, given alternatingly over each suprarenal region for a total of six exposures. The series was repeated two months later. The fall of hair commenced shortly after the first treatment and continued for several months. It is noteworthy, however, that the weight, size and arterial pressure remained uninfluenced by the irradiation. It is difficult to give a satisfactory interpretation of this partial result.

LEUCUTIA. [AM. J. ROENTGENOL.]

AN ANATOMIC AND PATHOLOGIC STUDY OF A SEVERE FORM OF ACRODYNIA. G. P. BELLOCQ and R. MEYER, Rev. franç. de pédiat. **8**:95, 1932.

The coordination between the clinical and the pathologic symptoms of this disorder led the authors to conclude that the principal pathology is a disorder of the neurovegetative system and the skin which it supplies. The asthenia is caused by lesions of the endocrine glands, particularly the thyroid. They believe that the primary cause of the pathologic condition is probably on an infectious or a toxic basis.

SANFORD, Chicago.

REPORT OF A CASE OF ACRODYNIA. LUIS MORQUIO, Arch. de pediat. d. Uruguay **3**:105 (March) 1932.

The condition occurred in a child aged 5 years. The case showed practically all of the classic symptoms of this condition. This distinguished and widely experienced clinician makes the statement that it is the first case observed in his country. The condition was first observed in Australia and then in the United States, and has since been reported with increasing frequency from many countries. The author points out the important rôle that infectious diseases seem to play in its etiology. The disease seems to be characterized by an encephalitic process and the development of a neurotropic virus which has a predilection for the mesencephalic region and causes considerable involvement of the sympathetic system.

SCHLUTZ, Chicago.

TEST OF SUSCEPTIBILITY TO ECZEMA IN PATIENTS WITHOUT CUTANEOUS ERUPTIONS, IN THOSE WITH ERUPTIONS AND IN ALLERGIC PERSONS: ITS PRACTICAL MEANING. ERNA ZITZKE, Dermat. Wchnschr. **95**:1425 (Oct. 1) 1932.

The author prepared saline extracts of seborrheic, psoriatic and normal human scales. These extracts were injected intracutaneously in dilutions of 1:10,000, 1:1,000 and 1:100. The test subjects included 65 noneczematous patients, of whom 26 had psoriasis or parasitic eruptions and 39 had normal skins, and 278 eczematous persons. In 13.8 per cent of the noneczematous and in 47.5 per cent of the eczematous patients the tests were positive with one or more of the extracts.

This indicates that 13.8 per cent of apparently normal persons are susceptible to eczema if exposed to relatively slight irritation and should avoid industries exposing them to irritating substances.

TAUSSIG, San Francisco. [ARCH. DERMAT. & SYPH.]

ASSOCIATION OF ERYTHEMA NODOSUM WITH TUBERCULOSIS AND WITH RHEUMATISM. NICOLAI JOHANNSEN, Jahrb. f. Kinderh. **131**:49 (March) 1931.

Erythema nodosum is an allergic disease of the skin, often caused by allergens originating in a tuberculous focus. Cases certainly occur which are not tuberculous; some of these are associated with polyarthritis. A full review of the literature is given.

CLAUSEN, Rochester, N. Y.

HISTOLOGY OF THE SKIN OF INFANTS, WITH SPECIAL REFERENCE TO THE CONNECTIVE TISSUE AND FAT. FELIZ WEISS, Jahrb. f. Kinderh. **135**:272 (May) 1932.

The skin of newly born infants, who were born at full term and were of low weight and vigor and of older infants, both healthy ones and those affected by nutritional upsets, was studied by the usual histologic methods. At birth, the differentiation of the epidermis of the soles is well advanced, but in other areas, it is not. The number of the nucleated layers is few; the granular and cornified strata are irregular, and the lamination of the latter is absent. The connective tissue portion of the corium develops steadily with age, but the elastic tissue is very slightly developed.

In "debility," the lobules of fat of the subcutis preserve their embryonic appearance; between the reticula are often seen islands of blood-forming tissue. In the infant born at full term, the fat stores of the soles are underdeveloped. This, together with the rich vascularity of the fatty tissue, explains the rosy color of the soles of normal infants. Mature development of the subcutaneous fat is attained only in late infancy. For this reason, various general noxae affect the subcutaneous fat of infants profoundly. The fat stores of the soles, however, are affected to the least degree.

CLAUSEN, Rochester, N. Y.

CONGENITAL DEFECTS OF THE SKIN. ILSE NORDMANN and JOHANNES SCHOEDEL, Jahrb. f. Kinderh. **136**:177 (July) 1932.

A case is reported in which there were various congenital anomalies that could be attributed to trauma which occurred during the fourth or eighth week of pregnancy. On the basis of this case and of others in the literature, it seems likely that few cases of congenital defect of the skin are due to defects of the ovum, to heredity, to criminal acts or to other forms of mechanical injury, but that most are due to a disturbance in the development of the amnion. Many other malformations formerly ascribed to defects in the ovum are attributable to amniotic adhesions.

CLAUSEN, Rochester, N. Y.

FEER'S FINGER-NAIL PHENOMENON IN INFANTS AFTER ERYTHRODERMIA. Z. BARABÁS, Orvosi hetil. **76**:15 (Jan. 9) 1932.

Feer's phenomenon, which is well known in connection with extensive desquamation after scarlet fever, is occasionally seen following erythrodermia. The infants have been followed in an institution very closely. About four or five months after erythrodermia the phenomenon usually appears, in the same form as after scarlet fever. The author explains it as a partial phenomenon of a desquamating process involving the whole skin. From the degree of development of the furrow on the finger-nail some conclusion can be drawn as to the intensity and duration of the original process.

ZENTAY, St. Louis.

Surgery and Orthopedics

UROLOGY IN CHILDREN. PHILIP GOLDFADER, Arch. Pediat. **49**:417 (July) 1932.

The author reminds one that modern urologic, diagnostic procedures employed in adults can be safely used in similar surgical urologic conditions in children. He further states that surgical conditions of the urinary tract are by no means uncommon at this age. Age is no longer a contraindication to proper surgical investigation and treatment. Methods of examination are described, and indications and contraindications are discussed. The various types of surgical, urologic conditions encountered in early life are discussed and reports of the cases illustrating some of these conditions are presented. WADDELL, University, Va.

OVARIAN CYSTS AND TUMORS IN CHILDREN UNDER TEN YEARS OF AGE WITH REPORT OF A CASE OF TERATOID CYST IN A CHILD FOUR YEARS OLD SIMULATING ACUTE APPENDICITIS. MARTIN J. LOEB and WALTER LEVY, Arch. Pediat. **49**:651 (Oct.) 1932.

A case of teratoid cyst in a child, aged 4 years, is presented. The picture was very similar to that of appendicitis. The writers review the literature of ovarian cysts in children under 10 years of age and discuss in detail the differential diagnosis of this condition. WADDELL, University, Va.

EXCISION OF HEMIVERTEBRAE FOR CORRECTION OF CONGENITAL SCOLIOSIS. EDWARD L. COMPERE, J. Bone & Joint Surg. **14**:555 (July) 1932.

When the hemivertebra interferes with the correction of the deformity, its removal will permit proper correction. The hemivertebra at the apex of the curve is usually the offending vertebra. Removal is followed by bone graft. The patient placed in plaster corset with a turnbuckle inserted, and correction of the deformity is gradually carried out. RYAN, Vancouver, B. C.

PREVENTABLE CHILD DEATHS: ACUTE APPENDICITIS. HENRY W. HUDSON, JR., New England J. Med. **207**:255 (Aug. 11) 1932.

The author first presents a graph showing that the deaths per hundred thousand from all causes in children between 1 and 10 years of age in Massachusetts had fallen from 1,280 to 322 from 1900 to 1930.

In 1900, the diagnosis of appendicitis was recorded 25 times and in 1930, 107 times, an increase of 428 per cent, while the child population had only increased 41 per cent. During these same years the death rate from appendicitis in the aforementioned age group increased from 5.2 to 15.8 per hundred thousand among the child population.

In the ten year period, from 1921 to 1930, there were 51,790 admissions to the wards of the Boston Children's Hospital, and the diagnosis of appendicitis was recorded 1,378 times, or once in each 37 admissions. Excluding admission for tonsillectomy and inguinal hernia, appendicitis was the most commonly recorded diagnosis. Experience shows that the policy, sometimes successfully followed in surgical conditions in adults, of observing the patient at intervals to determine whether the inflammatory process is increasing or decreasing, is folly in childhood; that perforation within from six to eight hours of the first symptom is not rare, and that early diagnosis, followed in most cases by early operation, is most essential in preventing childhood deaths from appendicitis.

Public instruction of the laity is urged to overcome the present notion that appendicitis rarely, if ever, occurs in childhood. GENGENBACH, Denver.

OPERATION FOR CLEFT PALATE. DENIS BROWNE, Brit. J. Surg. **20**:7 (July) 1932.

In this thorough article is a description of the anatomy and physiology of the palate. Based on these, the principles of the operation of cleft palate are worked out. The author argues that the mechanism is a sphincter, and the operation consists of the shifting of muscles to form an imitation sphincter, so that the normal mechanism of shutting off the nose from the mouth can be accomplished. The author advocates previous removal of the tonsils ,as they are a danger to the success of the operation. SUMMERFELDT, Toronto, Canada.

A CASE OF TRANSPLANTATION OF THE RIGHT URETER INTO THE CAECUM AND OF THE LEFT INTO THE SIGMOID FIVE YEARS AFTERWARDS. C. E. L. BURMAN, Brit. J. Surg. **20**:44 (July) 1932.

This operation is based on the advantages of the cecum acting as a reservoir for urine. In this operation the urine passes into a definite receptacle, and there is a less desire to pass urine as the absorption of water takes place the whole length of the bowel. There is no chance of solid contents interfering with the flow of urine, as the contents in this area are fluid. The cecum is easily fixed in a convenient position. The orifice of the stump of the appendix furnishes a convenient and comparatively easy and safe method of anastomosis by means of the inkwell method, and there appears to be no irritation when the urine passes into the cecum, as the acidity is quickly neutralized by the alkaline contents from the ileum. A case is cited illustrating the aforementioned points.

SUMMERFELDT, Toronto, Canada.

THE INTERVERTEBRAL DISCS: OBSERVATIONS ON THEIR NORMAL AND MORBID ANATOMY IN RELATION TO CERTAIN SPINAL DEFORMITIES. ORMOND A. BEADLE, Medical Research Council, Special Report Series, No. 161, London, His Majesty's Stationery Office, 1931.

The author has observed and summarized the results of the extensive researches on the spine by Schmorl at the Friederichstädler Krankenhaus in Dresden, where Schmorl removed and studied about 7,000 spines post mortem. Much attention is given to the development of the spine and, particularly, the intervertebral cartilage. The nucleus pulposus stands out especially distinctly in young persons, in whom it proves itself to be an organ in its own right. It is that part of the disk which plays the most outstanding rôle in pathologic changes. The author refers to the demonstration of fractures of the epiphyseal ring found in the macerated vertebrae which could not be demonstrated in the fresh specimen. It is rather startling to read: "By far, the most usual manner of origin of cartilage breaks is the action, not of coarse and violent trauma but of the imperceptible, ever active injurious influence of functional life working upon cartilages which are in some way inferior in their resistive power."

The senile changes in the cartilage are far more in evidence in any series of spines than the corresponding phenomena of adolescence, and are always more devastating in their effects. As a suggestive cause of the bulging of the cartilage into the bodies of the vertebrae, one finds a greater or lesser degree of osteoporosis of the spongiosa as a common senile change. The rupture and projection of the nucleus may also extend posteriorly, pressing on the posterior ligament. In a series of 368 spines examined, 56 (15.2 per cent) showed these formations posteriorly. They occur usually after fifty years, and affect the lower part of the dorsal and upper part of the lumbar region. Dandy described two cases of cartilaginous tumors in the lower part of the spinal canal, which he removed; these tumors had produced severe symptoms of disturbance in the nervous system. This monograph should be read in its complete form by all who are especially interested in the development of diseases and injuries of the spine.

PFAHLER, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

ABDOMINAL SYMPATHECTOMY FOR HIRSCHSPRUNG'S DISEASE. L. E. BARRINGTON-WARD, Proc. Roy. Soc. Med. **25**:122 (June) 1932.

The author gives first the history of sympathectomy and then the report of two cases of Hirschsprung's disease, in which he performed sympathectomy with gratifying results.

The photographs of one patient before and after operation are also given in the report.

WILLIAMSON, New Orleans.

IMPAIRMENT OF RESONANCE DUE TO APPENDICITIS IN CHILDHOOD. K. HOCHSINGER, Arch. f. Kinderh. **95**:223 (Feb. 5) 1932.

Normally, the percussion note is less resonant over the left lower abdominal quadrant than over the right, owing to the nearness of the descending colon and sigmoid. In the presence of symptoms suggesting either a chronic or an acute appendicitis, impairment of the percussion note on the right in the region of McBurney's point becomes significant. The anatomic basis is the inflammatory changes in the appendix, the cecum and the mesentery. The author feels that this aid to diagnosis is of particular value in children in which it is sometimes difficult to distinguish between voluntary and involuntary rigidity.

LESLIE, Evanston, Ill.

SCOLIOSIS. VON DETLEFF NEUMANN-NEURODE, Kinderärztl. Praxis **3**:109 (March) 1932.

The author, a physical therapeutist, lists the causes of scoliosis as follows: (1) tuberculosis and other infectious diseases, (2) late rickets and osteomalacia, (3) fractures of the vertebrae and their sequelae, (4) progressive muscular atrophy in its many forms, (5) unilateral muscle pull, due to hypertony, rickets and psychic or indefinite causes and (6) congenital anomalies of the spine.

Which of these, he asks, is susceptible of improvement by exercise therapy, namely, massage, passive motion and muscle stretching? Tuberculosis and other infections, acute rickets and fractures would be harmed by motion of any kind. Progressive muscular atrophy would not be influenced either way. There remains scoliosis due to muscular hypertonicity and congenital anomalies of the bony spine; the former is far more frequent. In the asymmetrical muscular hypertony, careful stretching of the spine will cause the scoliosis to disappear in from three to six weeks, if undertaken in the first year of life; the stetching will take from twelve to eighteen months if begun in the second year and the prognosis becomes very uncertain, if not hopeless, if undertaken after the third year. With congenital bony anomalies the stretching process lasts considerably longer. The stretching is accompanied by systematic exercise of all the muscles of the body, so that the asymmetry when corrected is not replaced by other asymmetries.

HUENEKENS, Minneapolis.

INFLAMMATORY DISEASES OF THE ORBIT IN OSTEOMYELITIS OF THE SUPERIOR MAXILLA IN INFANTS. E. CUSTODIS, Klin. Monatsbl. f. Augenh. **87**:631 (Nov.) 1931.

W. C. Posey was the first to describe the connection between diseases of the orbit and osteomyelitis of the superior maxillary bone in infants (Orbital Cellulitis from Disease of the Superior Maxilla in Children, *J. A. M. A.* **59**:1020 [Sept. 21] 1912). This type of osteomyelitis usually occurs during the first weeks after birth, and less frequently after the first year of life. In its most severe form it manifests itself by fever, vomiting and other general symptoms in addition to the maxillary condition. It is lethal within three or four days, even if extensive incisions are made. The second form shows the general symptoms in a less serious degree, the local changes predominating. Congestion and induration may extend from the lids to the cheeks and neck. The gingiva and alveoli are swollen;

abscesses and fistulas develop on the lower lid, especially at the nasal canthus, and finally the orbit and retromaxillary space become involved. The prognosis is unfavorable when metastases occur in the bones, joints, lungs and kidneys, and when toxic symptoms appear. The course in this type of disease may be acute or chronic. Four of the five cases observed by Custodis were lethal. According to Bosse-Meumann, who reported on three of Custodis' cases (*Arch. f. Ophth.* **126**:465, 1931), the mortality is 29 per cent. Puncture of the antrum with a heavy hypodermic needle is recommended for diagnostic purposes in advanced phlegmons. This may be done through the skin, the mouth or the nose. The initial stage of the disease is liable to be mistaken for a disease of the lids or tear sac. Subperiosteal progress of the disease may lead to displacement of the globe, and is usually coupled with protrusion and frequently with chemosis. Severe forms of phlegmons may involve the optic nerves or the pupillary tract, as happened in one of Custodis' cases, in which, furthermore, venous thrombosis caused preretinal hemorrhages. The expulsion of sequesters and the formation of encasing involucra may bring about recovery in milder forms running a chronic course. The therapy is surgical. Early and extensive opening of the diseased area is essential; splitting of the abscess alone is insufficient. Healthy tissues (especially the dental anlage) must be spared. Proper nutrition of the infant is of importance.

STOLL, Cincinnati. [ARCH. OPHTH.]

Miscellaneous

CHINESE CUSTOMS AND TRADITIONS OF CHILDBIRTH WITH BIRTH STATISTICS FOR SAN FRANCISCO. J. S. M. LEE, Am. J. Obst. & Gynec. **23**:434 (March) 1932.

This paper records some of the folklore of Chinese women in pregnancy and labor, few of which now obtain among the Chinese residents in San Francisco.

The Chinese methods of life in San Francisco do not affect unfavorably the maternal or fetal mortality. From Jan. 1, 1929 to Oct. 1, 1930, 649 Chinese women were delivered, with 3 maternal deaths (0.47 per cent) and 13 stillbirths (2 per cent). Of the 649 women delivered, 122 were delivered in the Chinese Hospital, with 1 maternal death and 4 children stillborn. The children in this series of 122 cases average 3,192 Gm. in weight.

These figures show that the Chinese in San Francisco are delivered with a maternal and fetal mortality that is not unfavorable to them when this section is compared with other quarters of the city. Improvement in mortalities will follow more adequate prenatal care, which now is limited not by racial customs but because of the expense.

ZIEGLER, Minneapolis.

DIAGRAMMATIC CHART SHOWING TIME OF APPEARANCE OF THE VARIOUS CENTERS OF OSSIFICATION AND PERIOD OF UNION. JOHN D. CAMP and EARL I. L. CILLEY, Am. J. Roentgenol. **26**:905 (Dec.) 1931.

The authors present a most comprehensive chart showing the normal development of roentgenologically important bones and epiphyses. The chart is unusually clear and should be most useful for teaching.

STULIK, Chicago.

CALCIUM AND THE TEETH. EDWARD PODOLSKY, Dental Cosmos **74**:780 (Aug.) 1932.

The author stresses the fact that in a large measure the presence of dental caries is due to an insufficient intake of calcium, which is characteristic of the American dietary. He recommends that calcium be taken in the form of large quantities of milk, fresh vegetables and also a calcium salt in the form of a gluconate of calcium. These, in conjunction with the judicious administration of vitamin D, the factor influencing calcium metabolism, will in large measure prevent the widespread malady of dental decay.

These precautions are practically essential during pregnancy, when the calcium reserves of the mother are drawn on. Should her calcium reserve be low, the bony structures and teeth forming within the fetus will be poorly developed, together with a weakening of similar structures within the parent. During the period of growth a proper amount of calcium therefore is also most essential.

MARKUS, Philadelphia.

BLOOD CALCIUM AND PHOSPHORUS FROM A DENTAL ASPECT. EDWARD C. DOBBS, Dental Cosmos **74**:867 (Sept.) 1932.

Dobbs presents data obtained from routine determinations of the calcium and phosphorus content of the blood in children under 3 years of age. He concludes that poor structure of the teeth and dental caries are associated with high blood calcium and low blood phosphorus. Further researches in the blood chemistry of children of different ages in various localities are urged to arrive at definite practical conclusions.

MARKUS, Philadelphia.

CARIES: A RÉSUMÉ OF OUR KNOWLEDGE OF ITS ACTION, TOGETHER WITH SOME OF THE MORE RECENT RESEARCH WORK. EDWARD H. HATTON, J. Am. Dent. A. **19**:1398 (Aug.) 1932.

Hatton briefly reviews the work of some of the men of several decades back on dental caries when this was thought to be due to acid formed by bacteria, the acid attacking the tooth surfaces. He then reviews the researches of present investigators who contend that caries has its origin from a lack of proper vitamins and insufficient minerals in the diet. The fact that the results of the latter show a considerable percentage of failures is sufficient evidence, Hatton claims, that the true cause has not been found. He is of the belief that eventually dental decay will be found to be due to some factor in the environment of the tooth acting on the tooth surface and having its origin in bacterial action, and not to nutritional disturbances.

MARKUS, Philadelphia.

SEXUAL DEVELOPMENT OF BOYS WITH SPECIAL REFERENCE TO APPEARANCE OF THE SECONDARY SEXUAL CHARACTERS AND THEIR RELATIONSHIP TO STRUCTURAL AND PERSONALITY TYPES. PAUL E. KUBITSCHEK, J. Nerv. & Ment. Dis. **76**:425 (Nov.) 1932.

Sexual development in boys is slow between the ages of 9 and 12 years. It becomes rapid at 13 and this rapidity lasts for about two years, when development slows down again. The primary sex characters are most marked between 13 and 14 years, the secondary characters developing more slowly and at a more uniform rate, with striking individual variations in the degree of this development. There is a definite relationship between the structural type and secondary sex character development, retarded development predominating among the asthenic types and advanced development in the athletic and pyknic types. There appears to be no particular relationship between structural and personality types.

These conclusions were drawn from a study of 730 boys ranging in age from 9 to 18 years.

JAHR, Omaha.

INFANT AND MATERNAL MORTALITY IN DENVER. F. P. GENGENBACH, J. Pediat. **1**:719 (Dec.) 1932.

A careful study was made of the infant and maternal mortality in Denver for an entire year, with follow-up studies for the ensuing year. Infant mortality was greatly higher in those cases in which there was no prenatal care by a physician and was appreciably reduced by more frequent visits to the physician during the prenatal period. The families with lower annual income showed considerably higher infant mortality than the families in better financial condition.

The mortality was very high along the South Platte river bottom, where the standard of living was found to be very low. This study indicates that decrease in infant mortality may be expected through education of people in better living conditions and in adequate prenatal care by physicians.

LEMASTER, Evanston, Ill.

MOTTLED ENAMEL IN ARIZONA AND ITS CORRELATION WITH THE CONCENTRATION OF FLUORIDES IN WATER SUPPLIES. H. V. SMITH and MARGARET CAMMACK SMITH, Univ. Arizona Col. Agric., Techn. Bull. no. 43, July 15, 1932.

Mottled enamel refers to a defect in permanent teeth in which the enamel has a dull chalky-white appearance and is frequently stained yellow-brown or black. This condition is frequently called "brown stain" or simply "stained teeth." Mottled enamel is found frequently in native-born inhabitants of certain parts of Arizona, as well as in other parts of the world, and has been proved to be due to the increased fluoride content of the water supply in these regions. In this study the authors analyzed chemically the water supply of practically every community in Arizona and also examined the permanent teeth of the school children for the presence or absence of mottled enamel. When the water contained less than two parts of fluoride per million, normal development of enamel was not disturbed. Concentrations of fluoride of from two to three parts per million produced variable results. The use of water containing over three parts of fluoride per million was always associated with mottled enamel. No correlation was found between the depth of the water supply and the fluoride content. It is suggested that individual susceptibility to this defect may also be affected by the nutritional condition, dietary deficiencies and fluoride content of the food supply, which are now being investigated.

LEMASTER, Evanston, Ill.

SCHOOL COOPERATION. F. JULIO PICAREL, Bol. d. Inst. internac. am. de protec. a la infancia **6**:129 (Oct.) 1932.

The nation is composed of manifold social cells which develop in different environments. These cells should promote all activities that are beneficial to society and, to such an end, join their efforts extending their good influence. But this will only be possible when a true spirit of cooperation shall possess all. Such a cooperation is necessary between the home and the school, a school made by and belonging to the people, so that its action may reach all the families in the form of joint and postschool activities: parent-teachers associations, children's leagues in and outside the school, temperance legions, Junior Red Cross societies, etc.

The practical objects of such cooperation might be to fight absentism, illiteracy and malnutrition; to buy teaching material, toys, food and books for the school library, and to promote the teaching of adults.

A Federation of School Cooperative Societies, constituting a powerful means of aiding in the solution of serious social problems, such as illiteracy, child welfare, workmen's education, etc., has been organized in Buenos Aires.

The author makes an appeal to all men of good will to go on working untiringly to improve the destinies of nations by means of a mutual understanding of the ideals of civilization and of reciprocal aid, uniting their honest brotherly hopes of a spiritual approach that may strengthen the bonds of human solidarity.

AUTHOR'S SUMMARY.

A SCHOOL FOR WEAK CHILDREN IN OBRAJES, LA PAZ (BOLIVIA). SATURNINO RODRIGO, Bol. Inst. internac. am. de protec. a la infancia **6**:147 (Oct.) 1932.

The National Antituberculous League of Bolivia started in December, 1930, the first School for Weak Children in Obrajes, La Paz. It is a model establishment possessing all the necessary conditions for boarding children who need a long

stay; another section is dedicated to day pupils, and a third is called the School Vacation Colony.

As the end is to insure the health and physical and mental development of weak children, the time allotted to instruction does not go beyond three hours a day. Preference is given to the developing of the senses and to drawing and manual work in the open air. Singing and recreation also receive great attention. The stay of the children at the school is never shorter than four months.

The staff is composed of a woman director, a medical counselor, a pedagogical adviser, a woman for recreation and gymnasium teacher, school teachers, nurses and domestic aids.

In a year and a half of work, 103 children, of which 44 were boys and 59 girls, of all school ages (between 6 and 14 years) left the school. Some remained more than four months and increased their weight (2 and even 3 Kg.) and their height; thus the school amply justifies the purpose which guided its creation.

AUTHOR'S SUMMARY.

SEX EDUCATION SHOULD BEGIN IN THE HOME AND CONTINUE IN THE SCHOOL. WALDEMAR COUTTS and GUILLERMO MORALES, Rev. chilena de pediat. **2**:383 (Aug.) 1931.

Fathers should be taught to develop the attitude that natural phenomena should not be presented to the child as mysteries and as moral errors.

Sex education should be obligatory in the school for both sexes. It should begin when the medical or physiologic counselor deems it opportune.

SCHLUTZ, Chicago.

ROENTGEN FINDINGS IN LEAD POISONING DURING CHILDHOOD. ERNEST KRAFT and KATSUJI KATO, Fortschr. a. d. Geb. d. Röntgenstrahlen **46**:249 (Sept.) 1932.

In three cases of lead poisoning in children dense subepiphyseal shadows were seen in the roentgenograms of the bones. The occurrence of these thick bands is an important aid in the diagnosis of plumbism. The lines in lead poisoning tend to be lamellated, while in phosphorus poisoning they are more sharply outlined and tend to be multiple. These findings were confirmed by experiments on animals.

LEMASTER, Evanston, Ill.

RELATION BETWEEN THE SIZE OF THE HOME AND SCHOOL REPORTS OF PUBLIC SCHOOL CHILDREN. ADOLF BUSEMANN, Ztschr. f. Kinderforsch. **39**:33 (Oct. 19) 1931.

Busemann found that there is a correlation between the size of the home (that is, the number of rooms and the number of dwellers in a room) and the school reports of the children.

The article is interesting in that it shows the extremely serious social conditions prevailing in the city of Breslau, owing to the fact that there is too little dwelling space for the population.

REICHLE, Cleveland.

THE ASSOCIATED MOVEMENTS IN PUPILS IN SPECIAL SCHOOLS. I. STEINMANN, Ztschr. f. Kinderforsch. **39**:83 (Dec. 15) 1931.

Normal children in public schools and children in special schools were examined to determine the frequency and type of associated movements. These movements were most frequently found in younger children and in both groups decreased with the age of the child. The intensity of the associated movement, however, was less in the normal child, particularly in the case of actions that are practiced early in life. The attempt to spread the toes was a good test for associated movements, which appeared in this case principally in the hands. In children

who show nervous defects and in those with organic disease of the nervous system intense associated movements are especially prone to develop. This is not a general rule, however, since a perfectly normal child in a public school showed the most intense associated movements seen in these groups.

REICHLE, Cleveland.

ROENTGENOLOGIC BONE CHANGES IN LEAD POISONING IN INFANTS AND CHILDREN. M. KASAHARA and H. HIROSHIMA, Ztschr. f. Kinderh. **53**:587 (Oct.) 1932.

The authors confirm the findings of Park and of Vogt that in chronic lead poisoning the ends of the long, growing bones show heavy, dense lines of calcification. These lines, however, are not distinguishable from those in elementary phosphorus poisoning or resulting after the administration of heavy metals. The finding of lead lines in the bones is an aid in the diagnosis of meningismus due to lead poisoning.

SHELLING, Baltimore.

REPORT OF THE DUTCH NATIONAL COMMITTEE OF ILLUMINATION ON ULTRAVIOLET RAYS. Nederl. tijdschr. v. geneesk. **76**:34 (Jan. 2) 1932.

In judging the effect of lighting one has paid attention only to that part of the light rays which gives the visible light. But in the future that part of the rays which is important for health must also be reckoned with. What are the qualifications of proper lighting? The Dutch National Committee of Illumination, department of the International Committee of Illumination, has asked some physicists and physicians to cooperate in determining the conditions which are necessary for ideal illumination. After extensive investigations, the committee came to the following provisional conclusions:

1. If artificial illumination is to satisfy hygienic conditions, one has to pay special attention to the spectral range of from 290 to 310 millimicrons. In this range the radiation must have such strength that its effect can be compared to that of the sun's rays in the same range.

2. The light of wavelengths shorter than those in the spectrum of the sun must be as faint as possible.

3. The source of light must be placed so that the direct rays will not strike the eyes.

In order to be able to formulate more rigid conditions in the future and eventually to arrive at a greater economy, it is necessary to observe the following rules: (*a*) The relation of wavelength to effect on the tissues must be determined in a large number of pathologic processes. (*b*) Special attention should be paid to the minimum dose of light necessary for the achievement of a favorable effect. (*c*) One must study the final effect of higher doses and the limits of the doses must be determined. (*d*) One must ascertain whether light with rays of other wavelengths has a detrimental or a useful effect; the doses at which these effects occur must then be determined.

As regards the irradiation necessary for the prophylaxis of rickets, the committee, after a careful study in which the results of recent investigations were closely considered, stated the opinion that much work must still be done on the quantitative side of the problem. Qualitative conclusions are as follows:

1. From sources of light to be used in prophylaxis of rickets (or therapy) should emanate rays of a wavelength of less than 310 millimicrons, and preferably no lambda rays of less than 280 millimicrons, for this light cannot have a specifically favorable effect; on the other hand, it probably prevents the desired forming of vitamin D.

2. A certain dose of irradiation given by means of weak rays applied for a long time may prove to be more efficient for the formation of vitamin D than the same dose when given by means of stronger rays applied for a shorter time, as the body can better withdraw the vitamin D from radiation coming from a greater distance.

VAN CREVELD, Amsterdam, Holland.

Society Transactions

AMERICAN PEDIATRIC SOCIETY

HUGH MCCULLOCH, M.D., *Secretary*

Forty-Fifth Annual Meeting, May 8-10, 1933, Washington, D. C.

OSCAR M. SCHLOSS, M.D., *President*

CLINICAL OBSERVATIONS ON 1,000 NEW-BORN INFANTS: A STUDY OF MORBIDITY AND MORTALITY. DR. ETHEL C. DUNHAM, New Haven, Conn.

The high mortality rate in the fetal and neonatal periods is well known, and the mortality rates in these two periods have shown little tendency to fall. Obviously any attempt to lower this rate necessitates an understanding of cases, a problem which has hitherto been attacked chiefly from the pathologic point of view. A clinical study was therefore undertaken in New Haven in January, 1928, with the object of studying morbidity as well as mortality from data based on current observations of a large number of infants and on the clinical records of both mothers and infants.

One thousand births were studied, 61 of which were stillbirths. The 939 infants who were born alive were examined at regular intervals, three times in the neonatal period, and once at or near the end of this period. A fifth examination was made after about one year to verify earlier observations.

The results of the study are based on clinical, historical and pathologic data. In all, more than 4,000 examinations were made, 665 of them when the infant was less than 24 hours old.

It was found that the causes of stillbirth could not be satisfactorily determined in the majority of cases and that in the neonatal period prematurity was by far the most important cause of death. An analysis of prenatal and natal factors which might influence fetal and neonatal morbidity brought out two main points: first, that prenatal care, when studied quantitatively and qualitatively, did not appear to lower fetal and neonatal mortality, and, second, that morphine given to the mother probably plays an important rôle in the delay of onset of the infant's respiration. This is of interest since the respiratory difficulty has hitherto been attributed to other influences, such as toxemia in the mother or trauma to the infant from operative delivery.

When morbidity in the neonatal period was studied it was found that in only 114, or 12 per cent, of the infants were no abnormalities of any kind found. Abnormalities, found in 825 infants, were classified in five groups and the conditions separated into those which were considered major and minor according to the effect on the infant's welfare or activity. It was found that, as would be expected, the minor far outnumbered the major conditions. Major conditions of the skin occurred in 6 per cent of the infants, injuries at birth in 9 per cent, infections in 10 per cent and congenital defects in 11 per cent. Other conditions, the largest group of which consisted of prematurity and immaturity, occurred in 34 per cent of the cases.

The data gathered from this series of 1,000 births give a more complete and accurate picture of the incidence of the usual diseases and abnormalities found in the neonatal period than can be obtained from study based on smaller groups of normal or abnormal infants. This study has made clear the need for further investigation of those aspects of the problem of fetal and neonatal mortality involving the relation of prenatal and natal conditions in the mother to the welfare of the fetus.

DISCUSSION

DR. WALTER LESTER CARR, New York: The subject that has been presented is important. A valuable contribution has been made toward an understanding of the causes of infant mortality. The medical profession has had a great deal of criticism from various organizations because there has been no appreciation of the causes of this mortality.

In going over the statistical records of 6,856 patients during a five year period at the Manhattan Maternity Hospital, I was led to believe that there were three factors which in a gross way could be considered in connection with the incidence of mortality and also of morbidity: (1) the mother, (2) the infant and (3) obstetric care. I found, for example, that the first birth had a great influence, i. e., the mechanical force of the mother, her anatomic structure, the type of labor and the incidence of disease. The infant influenced the problem by its position in utero, its size and the effect of toxemia or infection. The obstetric care and routine were important.

With the mother allowance had to be made for a first birth as compared with later births, not only because of the size of the pelvis, but because of mechanical factors and muscular control. With very large babies there were the risk of cerebral pressure and, later, the influence of venous stasis from delay in labor. The effect of toxemia cannot always be predetermined. The obstetrician is also a factor. It is unfortunate that many physicians regard forceps as the only cause of injury at birth. One must realize that many premature infants show damage. Those who have seen babies in obstetric institutions have noted that spontaneous deliveries and cesarean births have been followed by injury of one type or another, such as fractures and hemorrhages.

In regard to the morbidity of the new-born, I believe that there are fewer cases of jaundice when a clamp is used on the cord than when the cord is tied. In the treatment of atelectasis, there is less danger when there is an oxygen apparatus ready for use, instead of manipulation, which was formerly the usual method. When forceful manipulations have been persisted in to make babies breathe, hemorrhages have resulted. and hemorrhages have been seen in the suprarenal glands. Injection of from 25 to 35 cc. of mother's blood has had a good effect after delayed birth.

All in all, some mortality and morbidity can hardly be avoided. The injuries which are due apparently to manipulation may be found in part to be congenital defects which at the moment of birth cannot always be definitely diagnosed. The whole subject, however, is a matter for careful consideration and should be classified as has been done in the paper presented.

An institution may show a lessened infant mortality rate if the mothers are of a better class, even if they are hard working women. In the determination of all percentages one should have a fair judgment of the essential factors that influence mortality at birth.

One influence, a little outside the scope of this paper, is the study of the mortality of mothers. There is a common disregard for the mortality rate of women of the same age as mothers who die at the time of confinement. Frequently the figures of maternal mortality are noted as being unnecessarily high without a comparison with those for the mortality of women who are not pregnant. It is known that pregnancy increases the work of the kidneys, heart and other organs, but the mortality figures ascertained should not be ascribed to pregnancy alone during a stated age period.

DR, CLIFFORD G. GRULEE, Chicago: This paper, so far as my knowledge goes, presents one of the most exhaustive and detailed analysis or study of morbidity and mortality in new-born infants which has appeared.

There are one or two points which should be made. Usually analyses of this sort are divided into two groups; one, an analysis of a small group of patients under direct control, as in the hospital. Dr. Dunham's group is of this sort. The other is one of mass statistics of a large group of persons. I doubt very much if the latter will bear out her statement regarding the infrequency of intracranial hemorrhages.

I think that the statistics of Dr. Adair in Minnesota and of several others show that among general practitioners the incidence of intracranial hemorrhage in this country is much higher than is indicated by the hospital records.

Another point I should like to make is that the incidence of intracranial hemorrhage is higher in premature infants, so that it is difficult to separate the two groups.

DR. ETHEL C. DUNHAM, New Haven, Conn.: In relation to Dr. Grulee's comment, I should like to say that the infants studied were representative of three groups: private patients in hospitals, patients in wards and infants whose mothers were delivered in their homes by members of the hospital staff. It is obvious that this was a selected group. There would naturally have been an advantage in studying a larger group of unselected cases representing a whole community.

In regard to the low incidence of intracranial hemorrhage, I must point out that I included only those cases in which I felt the diagnosis was wholly justified, and therefore excluded some cases which others had called intracranial hemorrhage. This, however, brings up fine points of diagnosis which I cannot discuss at length here.

THE MANAGEMENT AND CONTROL OF JUVENILE DIABETES. DR. GEORGE B. BADER (by invitation), New York.

The diet of the diabetic child should be as nearly as possible like the diet of the normal child. This diet is high in carbohydrate and low in fat. From 2 to 3 Gm. of protein per kilogram of body weight must be allowed. The caloric distribution is, roughly, from 12 to 15 per cent of the calories as protein, from 30 to 35 per cent as fat and from 45 to 55 per cent as carbohydrate.

The question of the total amount of the food in calories in a specific instance is governed by the same variables as those which govern one in the feeding of the normal child. Enough must be given to permit normal increments of growth and gain in weight. Obesity must be avoided.

The fractional distribution of the diet was discussed. Two doses of insulin, sometimes three in the twenty-four hours, are necessary to maintain the patient sugar-free.

The reasons why the diet as described is advantageous are as follows: 1. It permits a well balanced diet, one which is adapted to the child's needs and tastes. 2. It does not differ from the diet of other members of the family. 3. It furnishes a factor of safety in uncompensated cases because of the excess of carbohydrate. 4. It may be a factor in the few reactions to insulin. 5. Fat depresses the efficiency of insulin. 6. The high carbohydrate, low fat diet improves tolerance. 7. The children are satisfied and do not steal food.

Specimens of urine furnish the guide for the dosage of insulin and readjustments in the diet.

Problems in management and control were briefly discussed.

The rôle of heredity in diabetes mellitus makes it a family problem, and the disease should be investigated from the standpoint of the family.

DISCUSSION

DR. SAMUEL Z. LEVINE, Flushing, N. Y.: It seems to me that in the final analysis, the criterion of a successful diet in diabetes mellitus is whether the diet will lower the requirement for insulin (raise the carbohydrate tolerance) or lower the tolerance for carbohydrate.

So far as I know, pathologically there is no evidence either way. In Dr. Bader's cases, the tolerance, except in one patient, was appreciably reduced. This may be the normal phenomenon in diabetes during the period of growth.

I wonder whether Dr. Bader has a comparable series of cases in which high fat and moderate or low carbohydrate diets were used to compare the dosage of insulin per total amount of dextrose tolerated, and, also, whether he analyzed the output of nitrogen in the urine as a measure of the protein metabolism. In children,

especially when growth is proceeding at a rapid rate, it is important to determine whether the child is maintained in positive or in negative protein balance.

DR. GEORGE B. BADER, New York: I have no comparisons, but I had hoped that Dr. Geyelin would be here, as he has some.

Smith and West of Los Angeles have put diabetic children on diets and have changed the carbohydrate and fat consistently, keeping the diets equicaloric. They found that insulin had more effect when the carbohydrate was high and the fat was low. Geyelin and Rabinowitz of Montreal and Adelsberg and Porges of Vienna have made the same observations.

THE FEEDING OF BANANAS AND OF SQUASH TO INFANTS AND CHILDREN. DR. HAROLD L. HIGGINS, Boston.

The diabetic child shows a better tolerance for banana than for most of the other forms of carbohydrate. After the tolerance for carbohydrate has been raised by the giving of bananas, other forms of carbohydrate may be successfully substituted. This action is similar to that frequently noted with levulose in diabetic patients.

Dried ripe banana (banana sugar) serves successfully as the added carbohydrate in the milk-water-sugar formula of infants, even of those under 1 month of age. Three premature infants were fed successfully on milk-water-banana sugar feedings.

The stools tend to become larger when banana is substituted for other carbohydrates. They are usually formed or semiformed, alkaline, of pasty consistency and without large curds or much mucus. Gastro-intestinal disturbances in babies on banana feedings are certainly as infrequent as in babies on other feedings.

With the large stools of the banana-fed infant, and possibly because of them, there is an increase of nitrogen in the stool. There is a corresponding decrease in the nitrogen in the urine. The retention of nitrogen is but little affected, when banana is compared with other sugars.

A large part of fecal nitrogen is bacterial. Possibly some of the benefit of the feeding of banana may be the diminution of bacteriolysis in the colon, with diminished absorption of bacterial nitrogen.

Squash contains large amounts of carotene. Assay of a highly colored, canned, cooked squash showed 0.2 mg. of carotene per gram of squash. One ounce of squash contains 6 mg. of carotene. Carotene is not the only yellow pigment of squash; there are also alcohol-soluble pigments, probably lipocarotenoids.

Infants were fed on formulas similar to the following one: milk, 15 ounces (444 cc.); cooked squash, 10 ounces (4,535.92 Gm.); cane sugar, ½ ounce (226.8 Gm); water, to make 30 ounces (887.25 cc). In this formula, 5 per cent added carbohydrate is two-thirds cooked squash and one-third cane sugar. Cooked squash contains 10 per cent carbohydrate.

The normally nourished infant on this formula will show definite yellow pigmentation of the skin (carotenemia) in one week. The sclerae remain clear. One child with keratomalacia (vitamin A deficiency) and one undernourished child showed a delay in the appearance of pigmentation of the skin.

The squash feeding is potentially rich in vitamin A. Children with vitamin A deficiency showed rapid clinical improvement both as to the ocular symptoms and as to the general nutrition. The feeding of squash leads to stools that are of a consistency similar to those of banana-fed children; the stools are quite yellow, a large percentage of the intake of carotene being excreted by the bowel. The infants fed the squash formulas did well nutritionally, usually showing excellent gains in weight. They were not immune to colds or to infections of the respiratory tract.

While the use of large amounts of squash in an infant's feeding is open to objection because the baby becomes yellow, small amounts should be of value in the routine feeding of infants.

DISCUSSION

DR. ALFRED F. HESS, New York: One hears most of the carotene preparations, or carotene in oil.

The past winter I have given forty children fairly large doses of carotene, 20,000 units a day, and in a large number carotenemia has developed, probably because of individual idiosyncrasies that are difficult to explain. If forty children in the second year of life are given carotene and are followed for a long period, as I did for a whole winter, or five months, carotenemia will develop in some of them and not in others. The carotene appears on the surface of the body to the greatest extent where the skin is thick. It is seen on the inner surface of the palms and not nearly so much on the outer surface, where the skin is thin. It is present markedly on the soles.

One can tell to what extent the blood will titrate for carotene by the discoloration of the skin. The depth of the yellow color of the skin runs parallel with the analysis of carotene in the blood. Vitamin A in the blood is almost absent in these cases, because the carotene is stored in the liver.

I have tried to find whether there is any pathologic change. The carbon dioxide combining power, the sugar in the blood and everything so far as examined have been normal. There is no irritation of the kidneys.

The yellow color of the skin passes off, as a rule, in the course of a few weeks. There seems to be some susceptibility to ultraviolet rays, so that children with carotenemia will have roughness of the face.

When there is a large amount of carotene in the skin, there may be a large amount of carotene in the stool. However, one cannot judge the appearance of the skin by the stool.

As Dr. Higgins has said, we have found no carotene protection against infection in regard to pneumonia in the institution. The same results are obtained when 20,000 units of haliver oil is given. In the course of five months, ending April 1, we have had as many cases of pneumonia, twenty-three in all, whether or not vitamin A was given.

There have been as many cases of severe and mild infections, pneumonia, infections of the skin, otitis media and conjunctivitis in the children who were given the provitamin, carotene, as in those who were given vitamin A in cod liver oil.

DR. KENNETH D. BLACKFAN, Boston: Do you have any information as to the mineral content of this new vegetable?

DR. HAROLD L. HIGGINS, Boston: I have no information regarding the mineral content of squash. It takes from two weeks to two months for the color to disappear completely.

The development of carotenemia in some babies and not in others probably depends on the absorptive ability of the intestines for carotene. Our figures showed that about 90 per cent of the carotene we gave by mouth appeared in the stool.

THE ACTION OF SODIUM CHLORIDE IN EDEMA PRODUCED EXPERIMENTALLY IN DOGS. DR. A. A. WEECH, New York, and DR. ELVIRA GOETTSCH (by invitation), Boston.

Selected experiments were reported dealing with the reactions produced by salt in dogs which exhibited varying degrees of hypoproteinemia.

Protocol 1.—A salt-poor but otherwise adequate diet was used. The plasma proteins were normal. After forty-six days 10 Gm. of salt was administered daily by gavage. There was anuria of twenty-four hours' duration, with a gain of 1 Kg. in weight. During the second twenty-four hours, there was a negative sodium balance, and the previous weight increment was partially lost. Thereafter no striking reaction was noted. The general level of the weight curve was slightly raised throughout the period of high salt feeding.

Protocol 2.—A protein-deficient, salt-poor diet was used. There was a gradual depletion of the plasma albumin. The theoretical weight curve, calculated from the known loss of nitrogen, early fell below the actual body weight, suggesting retention of fluid. This suggestion was confirmed by physical signs of pre-edema. After eight weeks of deprivation, when the plasma albumin had fallen to 1.7 Gm. per hundred cubic centimeters, a high salt feeding was instituted for eleven days.

The effect was not different from that observed in the normal animal. Three weeks later, when the albumin was 1.35 Gm., a high salt feeding caused a marked retention of sodium, a rapid gain in weight and generalized anasarca.

Protocol 3.—A protein-deficient, salt-poor diet was used. There was a gradual depletion of plasma albumin. The same early deviation of the theoretical weight curve from the observed weight curve and the same signs of pre-edema were noted. After eight weeks of deprivation, the plasma albumin being 1.1 Gm., a high salt feeding was instituted. The weight curve and sodium balance indicated a series of changing responses to the increased intake: (1) three days of salt equilibrium and stationary weight, (2) six days of positive salt balance during which the weight increased 3 Kg. and massive edema appeared and (3) seventeen days of negative salt balance during which the weight declined precipitously and the edema vanished. The diuresis was accompanied by a decrease in the plasma volume, as shown by increasing concentrations of albumin and globulin without a change in their ratio.

The following considerations give a partial understanding of the reactions encountered. The movement of fluid across the capillary is controlled by the algebraic sum of the mechanical and effective osmotic forces which surround its wall. These forces include oncotic pressure of the plasma, intracapillary blood pressure, oncotic pressure of the interstitial fluid and mechanical pressure of the intercellular spaces. A change in any of the forces in the direction which favors the formation of edema results in an increment in the mechanical stress which the intercellular spaces must withstand. The increasing stress which accompanies the early stages of depletion of plasma protein may be held responsible for the retention of fluid of this stage. The retention does not require a high salt intake, as it takes place slowly. Neither will a high salt intake produce any striking change in the rate of accumulation, for at this time the tissue spaces possess sufficient strength to limit the quantity of accumulated fluid. At a certain point, however, this strength fails; rapid accumulation becomes possible and occurs if abundant salt and water are provided. Suddenly raising the quantity of ingested salt may temporarily increase the filtration pressure and thus contribute to the breakdown of elastic tissue.

Although, in general, salt favors the retention of fluid, a large quantity may produce renal diuresis. The force of diuresis is sufficient to annul completely the tendency to retention of fluid caused by the low serum oncotic pressure. The complete effect of such diuresis on the balance of forces across the wall of the capillary is not yet known.

DISCUSSION

DR. IRVINE MCQUARRIE, Minneapolis: I should like to ask Dr. Weech whether he has data on the excretion of potassium throughout this study, particularly during the unusual period of diuresis.

DR. JAMES L. GAMBLE, Boston: I should like to ask Dr. Weech whether, according to his theories, the ingestion of salt and excessive water might be expected to set up a situation that would incite the kidney to diuresis.

DR. A. A. WEECH, New York: We have done very little in the way of measuring the excretion of potassium. In a few instances we found a steady loss of potassium throughout the experiment corresponding to the loss of nitrogen. This was regarded as additional evidence of constant breakdown of tissue. More than that, we have no information on the subject of potassium.

I believe that careful consideration is due the suggestion that salt in excess of water may have had something to do with this phenomenon. In other experiments we have given salt gavages in physiologic concentration and have obtained similar results; even in those instances in which hypertonic salt was given the animals were allowed free access to fluid. It was invariably observed that they drank copiously during the period immediately following a gavage. In this connection, however, our quantitative data are not sufficient for better analysis.

We do not have any observations on the total base excretion. In the charts as presented only the sodium balance was shown. We could equally well have

charted the figures for chloride balance, but in no instance was there any significant difference between the two.

We have not studied the relation between sodium bicarbonate and sodium chloride sufficiently to make a conclusive statement regarding this subject. So far, we have found no difference between the action of sodium chloride and that of sodium bicarbonate, provided the two are given in equivalent amounts.

RHEUMATIC INFECTIONS IN EARLY CHILDHOOD. DR. RUSTIN MCINTOSH, New York, and DR. CHARLES L. WOOD (by invitation), Washington, D. C.

From the records of the Babies' Hospital for the past twenty-five years we have assembled twenty-four cases of rheumatic infection with the onset of symptoms prior to the third birthday. These cases have been subjected to a mass survey, and tables are presented showing the symptoms and physical signs of the illness. Cardiac involvement was recognized clinically in all but two cases, and in one of these exceptions the heart at autopsy showed adhesive pericarditis and there were numerous Aschoff bodies in the myocardium, leaving only one noncardiac rheumatic patient in this early age group. Joint symptoms were complained of previous to admission in 46 per cent of the cases, but physical signs of arthritis were found in only 38 per cent during the period of hospitalization. No patient had chorea or subcutaneous nodules. Various other symptoms less specific of rheumatic infection occasionally occurred. The laboratory data were reviewed. Of the entire series, eleven patients died and ten were discharged improved and three unimproved. The six cases in which autopsies were performed were reported briefly.

DISCUSSION

DR. ALFRED F. HESS, New York: It might be of interest to know the age incidence of rheumatic fever in an institution that cares for normal children of this age. In the course of twenty years, in caring for children in an institution that harbors about 300 or 400 children from 3 to 4 years of age, we have seen only 1 with rheumatic fever. There have been none with chorea or pericarditis. It may be that we have missed such cases, but we have not noticed them while the children were in the institution or in the course of careful examinations at the time of discharge. It should be added that there has been no epidemic of scarlet fever during this period, although other infectious diseases have occurred.

I think that this incidence is in accordance with what Dr. McIntosh has found. Twenty years sounds like a long period, but it is not so statistically. The average group consisted of 300 children; the turnover was only about twice a year.

This shows that a different conception perhaps can be obtained from hospital experience, especially in a large city, than from a group of children in the same city drawn from a different class.

After all, this disease must not be considered as rare, although, perhaps, as Dr. McIntosh has said, it does not occur as frequently as has been supposed.

DR. HUGH MCCULLOCH, St. Louis: I have been interested in hearing Dr. McIntosh describe these cases, because my associates and I have had the same feeling and ideas he expresses in his report. We think that the disease is rare in this period of life only because it is frequently missed. It is recognized more frequently in institutions where adequate autopsy service exists, because so often the correct diagnosis is made only at autopsy.

Probably one reason why the condition is considered rare is because one looks for the manifestations of rheumatic heart disease that are ascribed to adults and to older children. I think that Dr. McIntosh's description of these symptoms should lead to a better recognition of the condition at this period of life.

There are several points which might be emphasized: 1. Small children tend to behave more like a nonsensitized person than like a sensitized or desensitized person. 2. The disease tends to be more widespread throughout the body, and the type of process tends to be less well defined. One finds numerous Aschoff bodies and rheumatic nodules, swelling of the joints, fever and other symptoms in

lesser degree than in the well defined types in older children. 3. The process in cases that terminate fatally is rapid. Usually the children are ill when they are examined and progressively get worse, until in a few weeks they die. 4. The tendency for the heart to be involved should also be emphasized. In attacks of rheumatic fever in adults the heart frequently escapes permanent injury, whereas in children the heart is the predominant seat of the lesion. A point that impressed us was that when edema was present it appeared to be more like the nephritic than the dependent type of congestive failure, such as is seen in advanced cardiac disease in adults.

The various points concerning the lack of evidence in the family history and the past history have been our experience also.

I might offer a suggestion concerning the lack of respiratory infections preceding the onset of heart disease. At the onset the initial infection may be very slight, and it has seemed to us that there is also a minor respiratory response. The degree of tonsillitis may attract little attention, and yet severe cardiac disease may appear shortly afterward.

Dr. Charles Hendee Smith, New York: In New York we have observed a number of children who have rheumatism in the first few years of life. Autopsy on a child 18 months of age showed advanced mitral disease. We have seen chorea in children 18 months of age. In addition to the children who have shown rheumatism in the first three years of life, a good many have come to us in the fourth year with definite heart disease of so long standing that it must have dated back to the early years.

I think that Dr. McIntosh pointed out an important matter one must not forget about rheumatism in young infants, that is, the fallacy of histories. Routine histories taken in hospitals cannot be trusted for detailed studies. An especially trained staff is necessary to obtain accurate ones. An example of this appeared in our study of pneumonia. In almost every textbook it is stated that lobar pneumonia appears out of a clear sky. By taking a careful history with trained workers, in connection with all the patients with pneumonia seen in 1931, we found that 98 per cent had preceding respiratory infections for from one to four weeks.

Dr. McIntosh did not mention two tests of importance: the sedimentation rate of red blood cells and the filament, nonfilament leukocyte count. In children of all ages with active rheumatism, the polymorphonuclear count is often not high, but the nonfilament count is almost invariably raised and the rise persists for a long time.

One girl, a private patient, recently had an acute attack of rheumatism during which she had pain in one joint for one or two days, perhaps a little pain in the back another day and no more symptoms. The total white blood count was elevated for a day or two but rapidly reached normal. The polymorphonuclear count was normal, but for six weeks she had a very high nonfilament count and a high sedimentation rate for about ten days.

Dr. S. G. Ross, Montreal, Canada: The presence of rheumatic nodules is sometimes of value in the diagnosis of this condition. I wonder whether Dr. McIntosh has any information as to the incidence of this sign in his cases.

Dr. Rustin McIntosh, New York: We had no patient in this series with nodules or chorea and none with epistaxis at the onset.

Blood Cells in Healthy Young Infants: I. The Leukocytic Picture During the First Three Months, with Special Reference to Hourly and Daily Variations. Dr. Alfred H. Washburn (by invitation), Denver.

This article will be published in full in a later issue of the American Journal of Diseases of Children.

Pseudohypertrophic Muscular Dystrophy in Three Brothers: Report of One Autopsy. Dr. Alfred Hand and Dr. Arthur D. Waltz (by invitation), Philadelphia.

Three brothers with muscular dystrophy, with an irrelevant history as to the parents, uncles, aunts and grandparents, were studied without anything new being

found as the result of laboratory examinations. The observations were as follows: The characteristic high creatinuria and the high urinary specific gravity were present; otherwise, the urine was normal. The Wassermann test of the blood was negative. Examination of the blood showed: urea nitrogen, 10, 12 and 16 mg. per hundred cubic centimeters, respectively; uric acid, 3, 3.2 and 2.4 mg., respectively; dextrose, 111, 90 and 98 mg., respectively. The basal metabolic rate of the oldest boy, the most helpless one, was —12, that of the next boy, who was still able to walk, was +11, and that of the youngest boy, the most active one, was +21. Blood counts were normal. The stools showed normal digestion of fats and proteins. Roentgenologic study revealed "a sella turcica smaller than the average for patients of these ages" but no other abnormality. Two of the patients died at the ages of 19½ and 15 years, and the other is living at 13½ years.

Autopsy was performed on the oldest one. Emaciation of the body and distortion of the spine were extreme. The skull cap was 1 cm. thick in places; the pituitary fossa was small, 4 mm. anteroposteriorly, 2 cm. broad and 7 mm. deep, but the pituitary body showed no changes microscopically. Studies of the brain, spinal cord, sympathetic ganglions and peripheral nerves made by Dr. Alpers revealed no pathologic findings other than a decrease in the number of cells in the lateral gray column, with distortion of the remaining ones and loss of much of the Nissl substance. The thymus measured 5 by 6.5 by 0.7 cm., and the Hassall corpuscles were large and hyaline, few nuclei being evident. The myocardium showed considerable fatty infiltration, and staining with scarlet R showed fat globules within the fibers as well as between them. The liver also showed great fatty infiltration; this was especially marked in the periphery of the lobules. The voluntary muscles were grayish to the naked eye; microscopically, they showed the customary changes of disappearance of many fibers through fatty degeneration, some of the remaining fibers being much larger than others, the former showing distinct striations, the latter having much fainter or even invisible striations.

The youngest of the three brothers had appendicitis last year, giving us the opportunity for biopsy of an abdominal muscle. Dr. Fowler of the Presbyterian Hospital made a microscopic examination and reported as follows: "There was replacement fibrosis with a few swollen muscle fibers here and there, some showing beginning necrobiosis, others retaining their striation. The perimysium showed a cellular increase."

For the past six weeks the patient has been receiving glycine, as advocated by Milhorât, Techner and Thomas, the initial dose of 1 Gm. being increased to the present one of 4 Gm. three times a day. It is too early to say more than that some definite signs of improvement have been noted, so that our hopes have been revived.

DISCUSSION

Dr. Oscar M. Schloss, New York: Thomas, Milhorât and Techner have reported promising results in the treatment of pseudohypertrophic muscular dystrophy by glycine. The results have varied in different cases; in some the improvement has been slight, and in others, very marked. Apparently very little change occurs in the size of the muscles, but in favorable cases muscular efficiency is much increased so that patients are able to execute movements which had previously been impossible. This treatment is comparatively new and must be used much more before one can come to any definite conclusions as to its efficacy.

THE TOTAL WATER EXCHANGE IN THE NORMAL INFANT. Dr. Samuel Z. Levine, Flushing, N. Y., Dr. M. D. Wheatley (by invitation), New York, Dr. T. H. McEachern (by invitation), Ann Arbor, Mich., and Miss E. Marples (by invitation) and Miss M. D. Kelly (by invitation), Flushing, N. Y.

Studies of the total water exchange of one normal infant under varying conditions of diet are presented, both for the physiologic interest of the results and as a means of illustrating concretely the application of the direct and indirect methods of measuring water balance. The former method is based on differences between

all sources of incoming and all sources of outgoing water; the latter method, on differences between the total body weight balance and the algebraic sum of the balances of organic substances, protein, fat and carbohydrate. The chief merit claimed for these studies is the determination by direct measurement of all the components involved in calculating the water exchange, thereby avoiding the assumptions heretofore found necessary. The strikingly parallel results secured in these observations by the two systems of measurement unequivocally establish their reliability.

The infant was studied during three experimental periods of approximately constant intake of fluid but successively normal, low and high caloric intake and during three comparable periods of constant caloric intake but successively normal, low and high intake of fluid. The data are of interest at present chiefly as they concern some of the physiologic mechanisms controlling the heat and water regulation of the infantile organism. Their practical implications in relation to disease will be considered at a later time.

The absolute amounts as well as the proportions of water lost through the different channels of elimination and the consequent retention of water within the body varied markedly with the different diets. The direct relationship which was found to exist between the size of the insensible compartment of water elimination and the caloric content of the diet and the closer parallelism between the urinary compartment and the intake of water imply that the primary functions of the two moieties of water loss, insensible and urinary, serve in the heat and water regulation of the body, respectively. The urinary compartment of water is apparently the most labile of all avenues of elimination; the loss of water through the skin and lungs is, on the other hand, seemingly under the control of a more stable mechanism.

The results obtained during the period of restricted intake of fluid were interpreted as indicating that neither an augmented production of heat within the body nor a retarded elimination of heat from the body through the physical agency of evaporation played a significant rôle in the causation of the fever induced in these observations by dehydration. The implication of an impairment of loss of heat in radiation and convection rather than an impaired evaporation as a factor in the pathogenesis of dehydration fever is sufficiently contrary to the views so frequently expressed that direct measurements of loss of heat through all avenues, preferably with the aid of a calorimeter, would appear to be imperative in an appraisal of the evidence. On the other hand, the results obtained during the recovery period of liberal intake of fluid strongly suggest that an augmented loss of heat in vaporization does aid in alleviating dehydration fever, when already present.

One final point which may be mentioned in this brief summary is that attempts to estimate the water balance from measurements of the inorganic elements, sodium and potassium, in the intake and excreta (urine and feces) were unsuccessful. Such attempts invariably resulted in estimated water balances which were appreciably higher than those actually secured by the two systems of measurement previously described. The discrepancies possibly may be explained by loss of these constituents in perspiration through the skin. These data, which unfortunately were not determined at the time of these observations, are at present under investigation.

DISCUSSION

DR. H. L. NEWBURGH, Ann Arbor, Mich.: It is a privilege to be permitted to talk before this group, of which I am not a member.

A few years ago Dr. Levine and I discovered that we were deeply interested in the same question but in a different order of human beings. He was dealing with infants and I with adults, so that we had an especially interesting time comparing results. It was gratifying to both of us to find that our information agreed unusually well. Each time we meet we expect to have a difference of opinion, but up to date we have succeeded in avoiding a serious argument.

One of the most interesting features to me is the difference between the effect of dehydration, or the difference between the sharp restriction of the intake of fluid, in the infant and in the adult.

Dr. Levine quickly produced fever, the usual, so-called dehydration fever, by restricting water. This fever was evidently not due to the lessened removal of heat by the vaporization of water; that factor was about the same as in the control period. There was some other mechanism involved.

We made similar studies on the adult. The restriction of fluid was, however, very much greater. Last year and the year before we studied a man whose total allowance of fluid was only as much as this infant received, so that, of course, the relative reduction was much greater. This man was up and about. The restriction continued for four days without any change in his temperature. So there apparently is an essential difference between the response of the baby and that of the adult to this process.

Similar studies with the adult dog did not produce fever. We have restricted fluid as sharply as possible for periods of five days without the development of fever in the adult dog. How the puppy would respond, I do not know.

Dr. Levine, of course, covered a great many features in this study, each of which is of intense interest to me. I think that it is hardly fair to take any more time to discuss the matter.

Dr. Irvine McQuarrie, Minneapolis: Our study on the effect of the state of hydration of the body on the insensible loss of weight has been confined largely to the intermediate age group. Fever was frequently produced at first in epileptic children between the ages of 5 and 12 years, following too rigid restriction of the intake of water. When attempting to control hydrocephalus in infants by the restriction of water we observed that dehydration fever develops in infants far more readily than in children of more advanced age.

While using Dr. Newburgh's modification of the Benedict and Root insensible loss method for determining the total energy metabolism in connection with our studies of the water balance in epileptic patients, we found the insensible loss per hour to be greatly reduced when our patients were subjected to marked dehydration, no matter what type of diet was being used. The degree of dehydration in our patients was evidently more marked than that of Dr. Levine's. In one instance the percentage of body heat dissipated by vaporization of water during a period of marked dehydration was less than half of the usual 24 or 28 per cent. At the same time, the total production of heat, as determined gasometrically, was little affected by the dehydrating measures.

On the other hand, the insensible loss of water was found to be greatly increased when a large positive water balance was established by the regular administration of antidiuretic pituitary extract during periods of forced drinking of water. In the absence of extremes in the state of hydration, however, estimations of the energy metabolism from the insensible loss per hour were found to correspond closely with values determined simultaneously by means of the Tissot method.

Dr. Fritz B. Talbot, Boston: Any one who has attempted to do such work as Dr. Levine has done appreciates the tremendous amount he has accomplished. It is creditable because it brings out so many facts of importance.

The correlation between the total production of heat, if generally applicable, is of great importance and will necessitate at least a rough estimate of the intake of food of all patients on whom the metabolism is being estimated, whether for diagnosis or for investigation.

The evidence presented is another step toward solving the question of luxus consumption and other problems connected with the intake of food.

Dr. Samuel Z. Levine, Flushing, N. Y.: The relationship between the energy metabolism and the caloric intake was based on the basal periods of the twenty-four hourly observations during which the infant was asleep. These periods totaled from about twelve to fifteen hours of the day.

The muscular activity was recorded from minute to minute throughout the experimental periods, thus affording a rough estimate of the effect of activity on the metabolic needs.

ECONOMY OF WATER IN RENAL FUNCTION REFERABLE TO UREA. DR. JAMES L. GAMBLE, Brookline, Mass., and DR. CHARLES F. MCKHANN, JR., and DR. A. M. BUTLER (by invitation), Boston.

The results reported were obtained from experiments on rats in which substances which must enter the urine were added to a basal maintenance diet. The urine was collected, and the concentration in the urine of the ingested substances measured. The intake of water was not restricted. The concentration values found in the urine are regarded as describing the optimal water requirement for the removal in the urine of the substances studied. The substances included a number of inorganic salts, sodium chloride, sodium bicarbonate, potassium chloride, potassium bicarbonate, potassium dihydrogen phosphate and disodic sulphate and the nonelectrolytes, creatinine, galactose and glucose (obtained by phlorhizinizing the animals). It was found that the salts, whether fed singly or as mixtures, produced approximately the same concentration value in the urine, and from this it is concluded that the water requirement for the removal of electrolytes in urine is the same for each of the component radicles of the salts studied and is additive for mixtures of them. When urea alone was fed it was found in the urine at a much higher concentration than the value established for the salts. When mixtures of urea and salt were fed, the concentration of urea plus salt in the urine was found to be much higher than the expected value derived from the values established for urea and salt when fed separately, indicating that when urea and salt enter the urine together their respective water requirements are much less than additive. The resultant economy of water was found to be almost one half of the expected expenditure. It was found that the other nonelectrolytes studied, creatinine, galactose and glucose, when fed with salt, do not exert this water-saving effect. When, however, they are fed with urea, an extensive reduction of the water expenditure below the expected level occurred.

TREATMENT OF CHOREA BY INDUCED FEVER. DR. CHARLES HENDEE SMITH, DR. LUCY PORTER SUTTON (by invitation) and DR. KATHERINE DODGE (by invitation), New York.

The results obtained in the treatment of 150 patients with chorea with fever induced by the intravenous injection of typhoid-paratyphoid vaccine were presented. For comparison, the cases of 150 patients treated in the same service prior to 1930, when the present method was started, were analyzed.

The cases in the two groups were divided according to severity into mild, moderate and severe. The therapeutic results were as follows.

The average duration of the chorea in patients treated by methods other than fever was:

Type of Case	No.	Average Duration, Days	Range, Days
Mild	49	27.4	10-67
Moderate	68	44.0	15-120
Severe	33	62.4	24-180

The average duration of the condition in patients treated with induced fever after fever treatment was started was:

Type of Case	No.	Average Duration, Days	Range, Days
Mild	68	5.72	2-14
Moderate	57	8.56	3-22
Severe	25	15.80	5-47

The average duration of the disease in all patients treated by this method was eight and five-tenths days. The average duration of the disease in all patients treated by other methods was forty-two and six-tenths days.

Physicians from various parts of the country have reported good results to us in about 60 other cases.

The advantage of this method is that the attack is definitely shortened, thereby saving expense to the hospital and decreasing the time lost from school. Children who have been treated during attacks and who have not been treated during attacks state that they prefer the treatment in spite of the disagreeable features.

The vaccine is given daily until the attack is over, unless the child seems worn out from a severe reaction. When this occurs a day's rest is given. A fever of at least 104 F. should be obtained to get the best results. It is important to remember that it is the fever rather than the means of producing the fever which is of benefit.

The vaccine used is New York City triple typhoid vaccine, which contains 1,000,000,000 typhoid bacilli per cubic centimeter and 750,000,000 each of paratyphoid A bacilli and paratyphoid B bacilli.

The usual first dose is 0.05 or 0.1 cc. undiluted, given intravenously. Subsequent doses are determined entirely by the febrile reaction of the child to previous doses. The average patient may take: first day, 0.05 cc.; second day, 0.075 cc.; third day, 0.15 cc.; fourth day, 0.25 cc.; fifth day, 0.4 cc.; sixth day, 0.6 cc., and seventh day, 0.85 cc. However, this routine cannot be counted on to produce the adequate elevation in temperature. A much greater increase may be necessary, or another dose on the same day may have to be given. This dose is usually from one-third to one-half the original dose of the day. For instance, if the first dose of the day is 0.6 cc. and at the end of two hours the temperature is 103.2 F. and fifteen minutes later is 102.8 F., then 0.2 cc. is given at that time. If the temperature has dropped below 102 F., 0.3 cc. is given.

Immediately after the injection the children are covered with blankets and are given hot water bottles to render the chill less unpleasant. Codeine relieves the headache. Minimum amounts of fluid are given, in order to reduce sweating, which tends to keep down the fever. No food is given within two hours of the injection, to reduce vomiting and nausea. If the temperature rises to 106 F. or more, the blankets are removed, acetylsalicylic acid is given and an ice-cap is applied to the head. If necessary, a cool sponge bath is given, or the child is put in a tub at 100 F. It has always been possible to reduce the fever when desired.

In an attempt to maintain the temperature for a longer period than is possible with vaccine alone, several children were put in tight blanket packs. Satisfactory fevers were obtained, and good therapeutic results followed. The method was abandoned because of one case in which after a sudden rise in temperature to 109 F. diffuse lobular pneumonia developed and the patient died. Autopsy also showed fresh vegetations on the mitral valve and encephalitis in the basal ganglions. The cause of death apparently was the pneumonia, which had not been suspected before the last treatment was given. No major complications have occurred from the vaccine alone.

Of the 150 patients treated, 28 already had organic heart disease. Eight had evidence of active carditis at the time treatment was started. None of these was harmed in any way by the treatment. The patients with active carditis lost the signs of activity by the time the treatment was over and the attack of chorea arrested.

From the results obtained by this method it is felt that an attack of chorea can be shortened to less than one fourth of the expected duration.

DISCUSSION

DR. E. L. BAUER, Philadelphia: Dr. Smith told me about induced fever therapy in chorea a little over two years ago, and I undertook it at the Jefferson Hospital with what I considered amazing success. Fortunately or unfortunately, I do not know which, I did not get all of the details. One of the details which I omitted was the codeine. I have never given codeine, and I have never regretted not doing so. Perhaps the children in South Philadelphia are a little tougher than those in New York, because I have never seen any of them vomit. With

their teeth chattering with chills, they say, "Are you sure I will be over this in time for dinner?" They do not lose their appetites.

I have not produced any excessive temperatures. While one or two just about touched 106 F. at the highest, I have never obtained anything above that. I have never resorted to packs or to any other effort to reduce the temperature except to let it come down as it would.

I have a report of twenty-three cases in press now. I did not report the cases of the forty-five patients treated to date because the earlier cases, I believe, would give some clue as to what happens to these children afterward, and the entire group might be confusing. None of the twenty-three patients has had a recurrent attack. A recent recheck has found them fully recovered. In the remaining twenty-seven cases there have been three recurrences. That is not surprising in view of the fact the infection is rheumatic, and rheumatism does recur.

However, some patients return with evidence of rheumatic fever without chorea, so that I have not felt at liberty to discharge them merely because this symptom has been arrested.

I believe that this problem will be one for hospitals for convalescents in time, because the rheumatism must be taken care of in the usual way, with a period of rest and the use of salicylates and gradually increased exercise, the heart being watched as a criterion.

We have had no increase or no particular decrease in the number of cardiac lesions as a result of this treatment, but I believe with Dr. Smith that it does reduce the time that the patients suffer from the acute phase of the illness.

Ten cases of chorea that I saw were so-called chronic chorea; the patients were ill for perhaps six months or longer. They all responded with one or two courses of treatment. I give eight febrile shocks on successive days, rest for eight days and then, if the choreiform movements have not ceased, eight more shocks.

Another observer in Philadelphia tried typhoid vaccine. He called me in to see a case in which he said treatment had failed. I realize the embarrassment one has in trying to define to the student choreiform movements. I found that he had a child with athetoid movements from another cause which he was confusing with choreiform movements. He did not, of course, get any results.

Incidentally, I like to see these children have chills as well as a rise in temperature. I believe that they react sooner if they have a mild chill; it does not inconvenience them greatly. They prefer the needle to the disease.

DR. JOSEPH BRENNEMANN, Chicago: I should like to ask Dr. Smith how long he keeps the children in bed after they are well. Are they carefully watched after they go home? Do recurrences ever appear within a short time after they go home? I should also like to know whether he has any information on the relative incidence of cardiac complications in patients so treated and in those no so treated.

DR. DEWITT H. SHERMAN, Buffalo: I should like to know whether there are any deleterious effects on the kidneys from the large ascending doses.

In one case of a small series at the Childrens Hospital in Buffalo, albuminuria developed and persisted during the patient's stay in the hospital.

Of our series of ten patients treated with triple typhoid vaccine, only five had good reactions; they improved as described by Dr. Smith. One of the remaining five patients was later vaccinated with malaria. The course of the chorea was shortened.

The reason we did not obtain as marked reactions as Dr. Smith is probably because we used 0.1 cc. rather than 0.25 cc. as the original dose; we increased the dosage by a ratio similar to his.

DR. BENJAMIN KRAMER, Brooklyn: The results that I should like to report were obtained by Dr. Solomon in our clinic. We had no such large series as Dr. Smith has reported. Nevertheless, there are certain facts which were brought out by our experience. We treated nineteen children. Twelve of them were boys and seven girls; the ages varied from 5 to 12 years; eleven cases were moderate

in severity and two very severe. Eleven patients were treated during the first attack. There was quite a difference in the length of time the patients had been ill before they were treated, anywhere from one week to one year. One hundred million organisms usually constituted the initial dose; this number was increased in some cases to as much as 800,000,000 for a single injection. The vaccine was given intravenously; eight injections usually constituted the series.

If no reaction developed, that is, if no chill or fever appeared within a few hours after the initial injection, another larger injection was given. Patients varied tremendously in their susceptibility to the vaccine, so that it was difficult to determine the initial dose. Two patients were refractory to 1,000,000,000 organisms.

In one case we obtained a temperature of 108 F., with severe chills and prolonged stupor. As a rule, improvement was first noticed after the fifth injection. In one case no improvement occurred in spite of two courses of treatment. The average stay in the hospital was eighteen days, which is considerably less than when other methods of treatment are used. At the time of discharge, the condition had improved markedly so that any one who did not know that the patients had had the disease would have failed to detect the residual signs of chorea.

In twelve cases there were very slight spontaneous movements at times; these were brought out only on attempted sustained voluntary activity on the part of the patient, or slight clumsiness developed when they attempted fine coordinated movements. We followed up thirteen of these cases. Three patients had no recurrence for eighteen months; two were followed for a year, four for six months and one for ten months. None of the thirteen patients had other manifestations of rheumatic fever while under observation. One patient had a recurrence of chorea after two months, and one after six months. In a third case progressive rheumatic heart disease developed, beginning with a systolic murmur; within four months there were signs of a double mitral lesion.

One patient with a very severe case was admitted to the hospital for vaccine therapy. While we were considering the advisability of giving this treatment, the patient's condition improved so much that we thought it unnecessary to give the treatment. Every one has had such experiences with Sydenham's chorea.

In general, we believe, therefore, that in very severe cases of chorea and in prolonged cases in which a preliminary period of rest in bed is of no benefit vaccine therapy is the best available treatment. It shortens the period of hospitalization and is the most effective treatment known at present. It certainly has definite advantages, as Dr. Smith has pointed out, over the use of phenyl-ethyl-hydantoin.

DR. CHARLES HENDEE SMITH, New York: We have had about 60 cases reported to us other than those added here, 45 of Dr. Bauer and Dr. Kramer, making 125 of which we know in which treatment was successful.

We have had the same experience that Dr. Bauer had; many of the referred cases were not cases of chorea. Dr. Dodge is working up the diagnosis of chorea largely from referred cases, such as those of athetoid, encephalitic syndromes, habit spasms and disease of the basal ganglions which simulates chorea.

The children with mild cases usually stay in bed a week, until they are thoroughly rested, and get up gradually, leaving the hospital within from ten days to two weeks. The ones with more severe reactions are kept in bed until they are rested and are more fit to go home.

We have a follow-up clinic where practically 100 per cent of the patients are examined; we are not talking about the effect on recurrences as yet, as it has been only two and a half years. We know that chorea recurs in a year, a year and a half or two years. Still it is our impression that the number of recurrences is less than in other groups.

Twenty-eight of the 100 patients had heart disease when they came to us, and the treatment did not do them any harm. Eight of them had acute carditis or signs of acute carditis, such as a murmur and enlargement of the heart suggestive of something more than a functional murmur, which disappeared. This condition may not have been carditis, but we thought that eight of the patients had severe cardiac involvement. We have had no case in which heart disease developed after treatment was begun. What this means we are not yet ready to say.

We have not seen renal complications. One investigator noted the appearance of hematuria following treatment, but we have examined the urine daily through the whole series and have never seen hematuria.

The variability of reaction is very definite. Some children are cured quickly, and others seem to require more vaccine and do not react quite so quickly. Our initial dose for mild cases is 100,250,000 bacteria. As the mild cases so often progress to moderate ones, it does not seem profitable for us to defer treatment. It is so much easier to cure the mild condition that it seems best to treat any patient who has a condition definitely diagnosed as chorea.

I wish to repeat the advice to keep the temperature elevated with hot packs. We did think that we could shorten the number of treatments in that way. I do not mean hot packs in the ordinary sense, but tight blanketing or the wrapping of each extremity with many layers of blankets to prevent all loss of heat. I have shown the total results on the chart. I might say that mathematically this treatment shortens the attack from one fourth to one fifth of the expected duration, that is, taking the mild, moderate and severe cases together.

SPREADING IN A MONOLAYER AS A METHOD OF STUDYING BIOLOGIC PROBLEMS. DR. E. GORTER, Leyden, The Netherlands.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

A STUDY OF THE TEETH OF A GROUP OF SCHOOL CHILDREN PREVIOUSLY EXAMINED FOR RICKETS. DR. MARTHA M. ELIOT (by invitation), DR. SUSAN P. SOUTHER (by invitation), DR. BERT A. ANDERSON (by invitation) and DR. SUMTER ARNIM (by invitation), New Haven, Conn.

The problem of the relation of rickets to defective formation of teeth and to the occurrence of dental caries has been approached through a study of the permanent teeth of 451 school children whose early history with regard to rickets was definitely known. The history of rickets was obtained from clinical and roentgenographic records kept in a previous investigation of the same children made during the years from 1923 to 1926. At the time of the recent dental examination, the children ranged in age from 5 to 12 years, the majority being from 6 to 8. One third of the group was colored, and two-thirds white. Of the 451 children, 215 were known by repeated roentgenographic and physical examinations to have had no rickets or only that of a minimum degree. One hundred and eight had shown evidence of moderate rickets by clinical examination but little or none by roentgenographic examination. One hundred and twenty-eight had had definite rickets of a moderate or severe degree as shown by roentgenographic examination.

The teeth of these three groups of children were investigated with special reference to dental caries and to hypoplasia of the enamel. A tooth was considered carious when there was cavitation of the exposed surface associated with necrosis and débris. The hypoplasia was characterized by a partial lack of enamel, the defects, as a rule, being symmetrically distributed in those portions of the enamel that were in the process of formation at the time of injury. The defects in the enamel were usually found in teeth in the process of formation, or during childhood, when rickets is prevalent.

Analysis of the data showed a definite relation between hypoplastic defects of the enamel of permanent teeth and rickets. The defects occurred considerably more often in children with a history of severe rickets (34 per cent) than in those with no rickets or rickets of a slight degree (4 per cent). Conversely, in practically every case in which severe hypoplasia was present, it was found that a history of moderate or severe rickets had been definitely established by roentgenographic examination.

Our data show that children who have had several of the severe infectious diseases of childhood (including pneumonia and bronchitis) are more likely to have defects of the enamel than children who have had fewer of these diseases. With

respect to the latter finding, it seems reasonable to suppose that when a severe infectious disease is superimposed on a rachitic process interference with the normal formation of enamel occurs more frequently and the injury is greater, than when no such disease is present. Whether the infectious disease causes the hypoplastic changes or whether the disease increases the disturbance of mineral metabolism already present and so indirectly produces the defects cannot be demonstrated from our data, but it seems that the latter view is the more probable one.

Moreover, from the data available, it was not possible to show the relation of hypoplastic defects to tetany. That a considerably higher incidence of convulsions occurred in children under 3 years of age with hypoplasia (22 per cent) than in those without hypoplasia (6 per cent) is of interest in this connection.

Caries occurred somewhat more frequently in the teeth of the children with a history of rickets (63 per cent) than in those who had none (43 per cent). It was also found more frequently in hypoplastic incisors and molars. The data on the latter point, however, do not show a causal relationship between hypoplasia of the enamel and caries, but suggest that the greater incidence of caries in hypoplastic teeth may account for its greater incidence in the teeth of children who had moderate or severe rickets.

That the administration of cod liver oil in infancy will reduce the incidence of hypoplastic defects and also to some extent lessen the incidence of caries was shown.

DISCUSSION

DR. F. F. TISDALL, Toronto, Canada: This paper is indeed interesting, because in Toronto we have been working somewhat along the line of Dr. Eliot, but our children are older. She has shown definitely some relation between antirachitic therapy and its effect on teeth during the developmental stage.

We have been interested in producing dental caries in animals, and have succeeded in producing and preventing it at will in rats.

The factors, under our experimental conditions, included an adequate supply of phosphorus and of vitamin D. When these two factors were present in sufficient amounts in the diets, caries was prevented; when these elements were reduced, caries occurred in 100 per cent of the cases.

Of course, we are not interested in the production of caries in rats. We are interested in the production of caries in human beings. We have for the past two years been carrying on an investigation in three orphanages, the children ranging in age from 5 to 15 years.

As you know, Dr. May Mellenby has shown that in England additional vitamin D lowers the incidence of caries, but English conditions are different from conditions in the United States. Those of you who have been in England during the winter months realize that one has to be a hardy person to survive the climatic conditions. I was there two months ago and have not recovered yet.

In our work we divided an institution into two parts, one half of the children being left on the prevailing diet, and the other half receiving only vitamin D in addition. The children lived under good conditions; they were in the country and got sufficient sunshine, when sunshine was available. They were on a diet which included 1 quart of milk, a fresh vegetable, some fruit and meat every day. In doing this work, we had the cooperation of five members of the dental profession. I shall show you some of their work in 2 or 3 slides to indicate how the presence of dental caries was determined. I may say that more than 6,000 x-ray pictures were taken.

I have been told by the dentists that it is extremely difficult, if not impossible, by ordinary dental examination to determine accurately whether there is decay or whether the old decay is stationary or progressive. The difficulty in detecting cavities by ordinary examination and the impossibility of getting an accurate picture unless an x-ray picture is included can be seen from these pictures. It is too soon to state the final results of these examinations. There are between 300 and 400 children under observation at the present time, and only about 150 have had their final examination. However, examination showed that the children who received

additional vitamin D under conditions comparable to your patients and mine had a definitely lowered incidence of caries.

I think this work is in line with Dr. Eliot's, and shows that antirachitic factors are also factors in the prevention of defects and decay of teeth.

DR. ALFRED F. HESS, New York: This work is interesting and timely. I have had an experience, though not as wide as that of Dr. Eliot, in connection with children in our institution in the last six or eight years in whom rickets has developed. We have followed up for two years about 45 children who definitely had rickets about six or eight years ago and also an equal number of children absolutely protected against rickets, as shown by clinical examination, examination of the blood and x-ray pictures. There is a great difference between these cases and those of Dr. Eliot, in that the rickets was mostly of a mild nature.

In New York City only about 15 per cent of children, so far as we can tell from x-ray pictures, have rickets, which should not be regarded as mild rickets, but is a moderate form. The cases Dr. Eliot studied were much more severe.

On examining the children, who are now between 6 and 8 years of age, we were able to note the deciduous teeth as well as the permanent teeth. We found a definite difference in the incidence of caries in deciduous teeth. There were perhaps twice as many cavities in the deciduous teeth of children who had rickets in infancy as in those of children who had been protected by cod liver oil, ultraviolet irradiation or some other agent.

When we studied the permanent teeth, we used the first molar, as did Dr. Eliot. There was just as much caries in the first molars of children who had been protected against rickets as in those of the children who had the mild form of rickets.

In perhaps two thirds of the children 8 years of age the teeth which are supposed to last a life time actually lasted only a year or two without undergoing disintegration. I think that the patients who have had tetany and those who have had rickets should be classified in two distinct groups: those with a low calcium content and those with a low phosphate content. The children who have had rickets for short periods, for instance, up to 6 months of age, those who have had it no longer than a year and those who have had it still longer should also be separated into groups.

Almost no calcium is laid down in the first molar tooth during the first six months of life, and practically none is laid down at birth. If the jaw of a newborn infant is dissected, the first molar will be found to contain no calcium, and it contains very little at 6 months of age.

In regard to the deciduous teeth, there was a difference between the incidence of caries among the children who were admitted to our institution the first six months, or from 6 to 12 months of age, and the incidence among those admitted later, from 1 to 2 years and from 2 to 3 years of age. We found much less caries among those admitted the first year. We attributed the difference partly to the fact that the children received an antirachitic substance, more especially cod liver oil, in the first year of life.

We carried out another test which might be of interest, especially as Dr. Eliot mentioned colored children. I have been connected with the preventorium for tuberculosis for a good many years; it has an equal number of colored and white children. These children come from New York City, from the district of Columbus Hill, where about fifteen years ago I made a study of rickets and found that practically all the colored children had rickets. I thought that it would be interesting to compare the incidence of caries in the permanent molars of 200 white children at this institution with that of 200 colored children who came from the Columbus Hill district, where, as I say, there was almost 100 per cent rickets at the time the children were born. I found that the teeth of the colored children were better in every way in each age group than those of the white children, in spite of the fact that the children were from the worst rachitic district in the city. When we compared them with white children of the same age from New York City, they were found to have definitely better teeth.

I must emphasize again that when I refer to the children with rickets in the institution I refer to the mild form. The children had no rickets as determined by x-ray pictures, merely a low phosphate content, or the kind of rickets that we all see among our patients and the type that is most common in present communities.

DR. HENRY J. GERSTENBERGER, Cleveland: Our experience in Cleveland has been like that of Dr. Hess. About six years ago the dentist at the Davis Dispensary said to me, "Why is it, Dr. Gerstenberger, that the colored children who have such severe rickets have better permanent teeth than the white children?"

DR. HAROLD C. STUART, Boston: It seems to me that Dr. Eliot's work offers an unusual opportunity for a study of growth, not only of teeth but also of the jaws and the face. It would be interesting not only to learn something about the growth of the jaws and bones of the face at the present time, and to know what effect rickets may have had on such growth, but also, if it is possible to follow the children, to determine what effect the defective teeth have on the ultimate development of the bones and on the eruption of the permanent teeth. If the x-ray films from the earlier period are still available, it seems worth while to review them from the standpoint of vitamin C deficiency.

DR. FRANK C. NEFF, Kansas City, Mo.: Dr. Eliot's contribution furnishes long-needed data on a group of children collected during the time they were known to have rickets and followed until their teeth appeared. It is only from the study of individual cases throughout such a period that one has a right to assume that hypoplasia may have been due to rickets.

DR. J. H. MASON KNOX, JR., Baltimore: I have been much interested in Dr. Eliot's paper and in the discussion. I wonder whether there are not factors concerned in the teeth of children other than the incidence of rickets. In the examination of rural children in Maryland we have found much more rickets, as indicated by changes in the bones on gross examination, in the colored than in the white children, but the teeth of the colored children have shown markedly less caries than those of the white children at corresponding ages. A partial explanation may be that the colored children are given hard foods that require chewing and a smaller proportion of liquid and soft foods at an earlier age. This may have something to do with the development of firm enamel and a greater resistance to caries.

DR. MARTHA M. ELIOT, New Haven, Conn.: The deciduous teeth of approximately 80 per cent of the children we studied showed caries, but the deciduous teeth of only 20 of the 45 children showed defects of the enamel. When we studied the defects in relation to the early history of rickets, we found there was a slight preponderance in the incidence of hypoplastic defects of the enamel among children who had severe rickets, but no difference in the incidence of caries.

With regard to the relation of caries and hypoplasia, I should like to point out again that though we found caries twice as often in the permanent first molars of white children as in those of colored children, the opposite was found to be true in the case of hypoplasia. Hypoplastic defects of the enamel occurred significantly more often in all types of teeth among colored children than among white children. The increase in hypoplasia among the colored children was shown to be closely associated with the greater amount of rickets in this racial group.

In reply to Dr. Stuart, I may say that we have made casts of the teeth of 250 of the children examined. We hope to repeat our developmental studies at the end of a year or two and to make examinations for defects in the enamel of the permanent teeth that have not yet erupted.

(To be concluded)

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

HARRY BAKWIN, M.D., *Secretary*

Regular Meeting, Feb. 9, 1933

JOHN CAFFEY, M.D., *Presiding*

THE TEMPO OF PHYSIOLOGIC DEVELOPMENT. DR. FRANZ BOAS.
(No abstract furnished.)

ROENTGENOGRAPHIC STUDY OF DEVELOPMENTAL GROWTH. DR. T. WINGATE TODD, Western Reserve University, Cleveland.

The roentgenographic study of human growth resolves itself into three subdivisions: studies, respectively, of maturity of tissue, of susceptibility of tissue and of adaptability of tissue, evidenced in three phases of growth, namely, from birth to approximately 3 years of age, from 3 to approximately 13 years of age, and during adolescence.

In a study of the maturity of tissue the adolescent phase is the least difficult to measure quantitatively. A criterion of quantitative determination of maturity being established (Todd, T. W.: Skeletal Growth and Development, in Growth and Development of the Child, White House Conference on Child Health and Protection, New York, Century Company, 1933, pt. 2, p. 26), the status of any adolescent can be assessed roentgenographically by the pattern of epiphyseal union, a method also applicable to other mammals. Progress in maturation can be determined by serial roentgenographic examination. When the assessments are checked against the standards they give definite measures of maturation hitherto lacking in health examinations, or at best merely indicated by such features as menarche, appearance of sexual hair, growth of genitalia and changes in the voice and in the velocity of the increment in the stature.

The progress of maturation is much the same in mediocre adolescents as that shown in the skeletons of dead adolescents, but in both these groups, it falls behind that of superior adolescents when mediocrity or superiority is qualitatively differentiated on a basis of constitutional health.

The increased velocity of dimensional growth precedes activity in the phase of maturation and continues, indeed accelerates, after the increment in the stature wanes.

Modifications of the pattern are characterized by imbalance between progress in physical growth, physical maturation, mental expansion and emotional stability.

In the phase of developmental growth, roughly covering the years from 3 to 13, maturity may be roentgenographically measured by the penetration of ossification into the epiphyseal cartilages (Todd, T. W.: Roentgenographic Appraisement, in Growth and Development of the Child, White House Conference on Child Health and Protection, New York, Century Company, 1932, pt. 4). Again the same relation between growth and maturation is seen as in adolescence; each occurs in active and quiescent phases, the precise age of expression depending on factors comprised in constitutional health. During this period the impacts of disturbance in health or progress manifest themselves in temporary delays rather than imbalances, so that maturation or growth, or both, fall behind schedule and can be restored by reestablishing metabolic integrity (Diabetic Children: General Considerations, in Growth and Development of the Child, White House Conference on Child Health and Protection, New York, Century Company, 1933, pt. 1, p. 351; pt. 2, p. 86).

In the phase of developmental growth comprised between birth and 3 years of age the maturity can be indirectly assessed roentgenographically by a study of epiphyseal ossification and diaphyseal dimensions and texture (Growth and Development of the Child, White House Conference on Child Health and Protection, pt. 2, pp. 52, 53 and 92). During this earliest phase of developmental growth the impact of disturbances in health produces a far more serious interruption of progress which amounts, in fact, to an inhibition rather than to a delay—an inhibition from which the child may, in the more severe grades, never adequately recover.

For the second caption of the study, namely, the susceptibility of tissue, the diabetic children mentioned earlier form an appropriate introduction. Treatment with insulin restores metabolic integrity and, thus encouraged, the pattern of growth reestablishes itself. One should look philosophically for promoters of growth rather than for stimulants to growth.

The so-called transverse line or scar on the bone shaft is a measure of constitutional disturbance, and the duration of its persistence gives an indication of the severity of the set-back. Scars are relatively infrequent under 3 years of age because the constitutional response is that of inhibition rather than that of delay. They rarely occur after 13 years because there is relatively little physical constitutional set-back.

During infancy a severe disturbance of the pattern of growth from which adequate recovery is impossible is registered in local areas of which the face is one (Todd, T. W.: Hereditary and Environmental Factors in Facial Development, *Internat. J. Orthodontia* **18**:799, 1932).

The third evidence of growth, namely, the adaptation of tissue, is appropriately illustrated in the physical features of the allergic skeleton, for the secondary or mobile calcium, fluctuating with the potential or expressed exudative diathesis, gives clear indications of the constitutional condition and measures the progress of affliction and recovery.

The fluctuations of the mobile calcium depot give a clue to other constitutional conditions, as can plainly be seen in serial roentgenograms taken during pregnancy and during adolescence.

DISCUSSION ON PAPERS BY DRS. BOAS AND TODD

DR. C. W. CRAMPTON: The two addresses impress me with the value of successive periodic records of a person. This principle, logically carried out, calls for periodic records made on the same subject during the entire extent of his life. We need life records. With life records we can make progress toward life diagnosis, life therapy and life management. Life records are to be the basis of much significant progress in medicine, for it is recognized that disease is often a matter of decades and centuries and not a matter of the moment. Almost nothing in medicine is sudden, not even apoplexy, and I believe that the future may demonstrate that little in medicine is unpreventable. When we have life records, we shall know the courses and byways of diseases which we now know only in the grossly evident final stages.

The speakers were good enough to refer to the principle of physiologic age and my labors in the subject as long ago as the beginning of this century. I may be pardoned a certain satisfaction in noting a continuing and increasing interest in the matter.

Thirty years ago, by giving a person two examinations annually for three or four years, I was able to follow the changes in the growth and development. For this reason it was possible to state that accelerations in growth and developmental changes were concurrent and were influenced by the season, and that developmental epochs were more markedly independent of chronological age than the purely generalizing methods of handling statistics revealed or, in fact, could reveal.

This points only to the fact that successive annual and semiannual examinations by competent scientists, not for the few years of adolescence but throughout the

entire life, promise to bring on the golden age of rewards and merits in the management of human lives. I believe that this scientific section can do no better than to address itself to a program of bringing this about.

The work is well under way. The Boy Scouts, the Girl Scouts, the Camp Fire Girls, the 4-H Clubs, the Y. M. C. A., the Y. W. C. A. and the schools constitute a group of 25,000,000 persons under the influence of organizations which have done something in this field and show promise of doing more. The aid of the pediatrician would be most welcome in this field.

DR. MYRTLE MCGRAW: I was interested in the patient whom Dr. Todd demonstrated as being accelerated in psychomotor though retarded in physiologic development, and notably deficient in calcium content. If Dr. Todd is using the familiar tests and scales for measuring the development of infant behavior, I doubt that such measures are adequate to justify his statement. The facts may be true, but my experience with tests and scales for infants is that they are at best rough measures and cannot be relied on to give a true picture of the development of behavior in a particular person. It is possible that Dr. Todd has devised other means of measuring psychomotor development in infants. If so, this criticism is unwarranted.

Book Reviews

Les feuillets du pédiatrie. By Dr. Germain Blechmann. Second series. Price, 16 francs. Pp. 232. Paris: Gaston Doin & Cie, 1933.

"The Pages of Pediatrics" represents the desire of a veteran practitioner to give the benefit of his experience to young physicians and to discuss the diagnosis and treatment of diseases of infants and children from a practical clinical point of view not adequately covered in textbooks. The book consists entirely of case reports on patients seen by the author, with comments which he deems it advisable to emphasize in each instance. These chapters were first published in *l'Hôpital* and *Le concours médical* and later collected in book form. The conditions discussed are the common and varied problems to be met in the office and home as they are taken from the author's records of his practice.

In some cases the fine points of bedside diagnosis and differential diagnosis are explained. In all cases general management and prescriptions for drugs, diet, physical therapy and other treatment are described with care. The author's impressions not only of the physical examination, but also of the mental attitude of the patient and family are interesting. Undoubtedly, a great deal of practical value can be obtained from a study of the daily records and observations of a practitioner of experience and judgment.

L'hygiène sociale de l'enfance. Volumes I and II. By Dr. G. Banu. Price, 100 francs. Pp. 1,700. Paris: Masson & Cie, 1933.

The social hygiene of infancy is presented in this publication in a most comprehensive and well organized manner.

In volume 1 eugenics and heredity are first discussed. Maternal hygiene is thoroughly considered from every angle. The greater part of the first volume is taken up with the physiology, feeding and general hygiene of early infancy, with one section on the infant mortality in different countries.

Volume 2 deals with the hygiene of preschool and of school children. Preventive medical measures, morbidity and mortality, food, physical development, behavior, special classes for handicapped children, legislation affecting schools and organizations for child care are only a few of the topics ably and completely presented.

The author has maintained an international point of view throughout, and has consulted the literature of many countries extensively. The entire work is exceptionally well organized. This publication should prove of the greatest value to persons interested in the hygiene and social aspects of children in every country.

Die intrakutane Kaninchenmethode zur Auswertung von Diphtherie-Toxin und Antitoxin. By Claus Jensen. Pp. 211, with 31 illustrations. Copenhagen: Levin & Munksgaard, 1933.

While studying the effect of the immunization of children with diphtheria anatoxin in 1927, the author saw the need of a practical and reliable method for the determination of small amounts of diphtheria antitoxin in the blood. Ehrlich's classic method was not applicable to this work. The results with Romer's technic on guinea-pigs proved unsatisfactory. The intracutaneous procedure on rabbits proposed by Fraser and Wigham in 1924 was found to give the most favorable results, to be the most exact, rapid and least expensive and to be suitable for the determination of extremely small, as well as very large, amounts of toxin by a single technic. The author has further developed this procedure and intensively investigated all modifying factors and sources of error, and has perfected the

method to a high point of accuracy. In this monograph these researches are described fully, and the directions for carrying out the tests are explained in minute detail.

L'acidose et l'insuffisance rénale aiguë chez le nourrisson. By Max M. Lévy. Price, 15 francs. Pp. 100. Paris: Masson & Cie, 1927.

The etiology and mechanism of acidosis are discussed from the theoretical point of view, with special regard to the ideas of Chabanier and Lobo-Onell. It is claimed that the most reliable index of acidosis is the ratio between the chlorides of the erythrocytes and the chlorides of the plasma. When the ratio is over 0.5, acidosis is indicated and sodium bicarbonate solution is administered. Several cases are cited to show the value of this ratio and its application in determining the therapeutic measures to be instituted. The author also discusses the physiopathology of acute renal insufficiency as it occurs in infants from excessive vomiting or diarrhea. The impaired renal function is attributed to an abnormal excess or decrease in tissue chlorides. The determination of the chlorides of the red blood cells furnishes the information necessary for proper therapy. If the chlorides are high, sodium bicarbonate solution is given. If the chlorides are low, salt solution is indicated.

The Tides of Life. By Dr. R. G. Hoskins. Price, $3.50. Pp. 352, with 6 illustrations. New York: W. W. Norton & Company, Inc., 1933.

The fascinating story of endocrinology will captivate the lay reader and the physician as well. The tremendous enthusiasm of the author for his subject permeates the entire volume. Each of the endocrine glands is discussed in a separate chapter. Enough of the early history of investigations in endocrinology is presented to give an adequate evaluation of the enormous advances in the knowledge of this field in recent years. Care has been observed always to keep within the bounds warranted by reliable scientific research, and the suggestions of the untold possibilities of the glands in influencing every-day activity, emotions and personality are discussed with judgment and caution. The basic anatomy, physiology and pathology of the endocrine system, as well as animal experiments and clinical observations, are presented adequately. The present-day knowledge of the endocrine glands and hormone activity is well summarized in an excellent, readable form.

Physiologie des Kindesalters. Volume II. By E. Helmreich. Price, 28.60 marks. Pp. 434. Berlin: Julius Springer, 1933.

A knowledge of physiologic processes and their normal variations during infancy and childhood is fundamental for the intelligent practice of pediatrics. In this book the growth and development of the child as a whole and of the various systems and organs of the body are completely discussed. The contents are divided into the following sections: growth, the osseous system, the musculature, the glands of internal secretion, the nervous system, the organs of the special senses, immunologic reactions, the skin and heredity. The subject matter is presented in an excellent manner. This volume is highly recommended to every physician who deals with children.

Clinique médicale des enfants: Maladies infectieuses. Deuxième Série. By P. Nobécourt. Price, 60 francs. Pp. 450. Paris: Masson & Cie, 1933.

This book is a continuation of a previous one, and contains clinical lectures on the different phases of measles, whooping cough, pneumococcic infections, streptococcic infections, erysipelas, infectious purpura, agranulocytosis, purulent meningitis, meningococcic meningitis, infantile paralysis and malaria. Each lecture has been published before in one of the French journals, and each is well illustrated with clinical cases. The book is useful from the standpoint of the clinician. The subjects are practically handled.

The Organs of Internal Secretion. By Ivo Geikie Cobb. Fourth edition. Price, $3.50. Pp. 303. New York: William Wood & Company, 1933.

This book on endocrinology was written for the practitioner of medicine. Considerable emphasis is placed on the clinical recognition of mild disorders of the endocrine glands, and hormone therapy is considered in detail. This edition contains new chapters on obesity and infantilism. There are not many references to recent work done by American investigators. At times the author does not discriminate clearly between pleuriglandular products of uncertain value and preparations of scientifically proved effectiveness.

Chinin in der Allgemeinpraxis. By Dr. Med. Fritz Johannessohn. Pp. 176. Amsterdam: Bureau tot Bevordering van het Kinine-Gebruik, 1932.

This book has been published in succeeding editions by the Dutch Bureau of the Quinine Industry. It contains a history of the subject and the various uses to which quinine is put. It is quite thorough; 465 references to the literature are quoted. For one who wishes to use quinine and to know about it, the book will prove useful.

Directory of Pediatric Societies

INTERNATIONAL

International Association of Preventive Pediatrics

President: Professor Taillens, Lausanne, Switzerland.
Secretary: Dr. P. Oltramare, rue Decandolle, Geneva, Switzerland.

International Pediatric Congress

President: Dr. George Frederic Still, 28 Queen Anne St., London, W. 1.
Secretary: Dr. Leonard K. Findlay, 61 Harley St., London, W. 1.

FOREIGN

Royal Society of Medicine, Section for the Study of Disease in Children

President: Dr. Leonard G. Parsons, 58, Calthorpe Road, Birmingham, England.
Secretary: Dr. W. G. Wyllie, 33, Wimpole St., W. 1, London.

NATIONAL

American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman Dr. Alfred A. Walker, Highland Plaza Apartments, Birmingham, Ala.
Secretary: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Pediatrics

President: Dr. Samuel McC. Hamill, 1822 Spruce St., Philadelphia.
Secretary: Dr. C. G. Grulee, 636 Church St., Evanston, Ill.

American Child Health Association

President: Dr. Samuel McC. Hamill, 1822 Spruce St., Philadelphia.
Secretary: Dr. Philip Van Ingen, 125 E. 71st. St., New York.

American Hospital Association, Section on Children's Hospitals

President: Dr. H. C. Schumm, 425 E. Wisconsin Ave., Milwaukee.
Secretary: Miss Sophie Yoerg, 721 N. 17th St., Milwaukee.
Place: Milwaukee. Time: Sept. 11-16, 1933.

American Pediatric Society

President: Dr. Charles A. Fife, 2104 Spruce St., Philadelphia.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid, St. Louis.
Place: Asheville, N. C. Time: May, 1934.

Canadian Society for the Study of Diseases of Children

President: Dr. Frederick F. Tisdall, 170 St. George St., Toronto, Ontario.
Secretary-Treasurer: Dr. T. G. H. Drake, 67 College St., Toronto, Ontario.

---

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Society of Pediatric Research

President: Dr. Rustin McIntosh, College of Physicians and Surgeons, Columbia University, New York.
Secretary-Treasurer: Dr. Charles F. McKhann, Harvard Medical School, Boston.
Place: Atlantic City, N. J. Time: May, 1934.

SECTIONAL

Central States Pediatric Society

President: Dr. Julius H. Hess, 104 S. Michigan Ave., Chicago.
Secretary: Dr. A. H. Parmelee, 715 Lake St., Oak Park, Ill.
Place: Chicago. Time: Sept. 22-23, 1933.

North Pacific Pediatric Society

President: Dr. Herbert E. Coe, 509 Olive St., Seattle.
Secretary: Dr. Raymond H. Somers, Cobb Bldg., Seattle.

Southern Medical Association, Section on Pediatrics

Chairman: Dr. Hugh L. Dwyer, 406, W. 34th St., Kansas City, Mo.
Secretary: Dr. Luther W. Holloway, 117 W. Duval St., Jacksonville, Fla.
Place: Richmond, Va. Time: Nov. 14-17, 1933.

STATE

Alabama Pediatric Society

President: Dr. C. E. R. Parker, 401 S. Court St., Montgomery.
Secretary-Treasurer: Dr. John W. Simpson, 1117 S. 22d St., Birmingham.

Georgia Pediatric Society

President: Dr. Joseph Yampolsky, 311 Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N. E., Atlanta.
Place: Atlanta. Time: October, 1933. Augusta. Time: May, 1934.

Louisiana State Pediatric Society

President: Dr. Cecil O. Lorio, 245, 3d St., Baton Rouge.
Secretary-Treasurer: Dr. Maud Loeber, Tulane University, New Orleans.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. Adolph G. De Sanctis, 12 E. 11th St., New York.
Secretary: Dr. George M. Retan, 459 James St., Syracuse.

Nebraska Pediatric Society

President: Dr. W. O. Colburn, 724 Sharp Bldg., Lincoln.
Secretary-Treasurer: Dr. C. G. Weigand, 1614 Medical Arts Bldg., Omaha.
Place: As announced by committee. Time: Second Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

North Carolina Pediatric Society

President: Dr. J. Buren Sidbury, 15 N. 5th St., Wilmington.
Secretary: Dr. E. K. McLean, 121 W. 7th St., Charlotte.
Place: Various children's hospitals. Time: Twice a year.

Oklahoma State Pediatric Society

President: Dr. Clark H. Hall, 117 N. Broadway, Oklahoma City.
Secretary: Dr. M. J. Searle, 108 W. 16th St., Tulsa.

South Carolina Pediatric Society

President: Dr. J. I. Waring, 77 Rutledge Ave., Charleston.
Secretary-Treasurer: Dr. Julian P. Price, Medical Bldg., Florence.

Tennessee State Pediatric Society

President: Dr. Owen H. Wilson, 706 Church St., Nashville.
Secretary: Dr. Joe T. Smith, 511 W. Church Ave., Knoxville.

Texas Pediatric Society

President: Dr. Sidney R. Kaliski, Moore Bldg., San Antonio.
Secretary-Treasurer: Dr. Frank M. Martin, 321 Medical Arts Bldg., San Antonio.

Virginia Pediatric Society

President: Dr. J. B. Stone, 2042 Park Ave., Richmond.
Secretary-Treasurer: Dr. Thomas D. Jones, Medical Arts Bldg., Second and Franklin Streets, Richmond.

West Virginia State Medical Society, Section on Pediatrics

President: Dr. R. C. Hood, 428 W. Pike St., Clarksburg.
Secretary: Dr. Russell C. Bond, 1325 Chapline St., Wheeling.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. C. W. Burhans, 14805 Detroit Ave., Lakewood, Ohio.
Secretary: Dr. J. A. Garvin, 2009 Adelbert Road, Cleveland.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. Gladys Boyd, 56 DeLisle St., Toronto.
Secretary: Dr. Lawrence Murray, 1870 Bloor St., W., Toronto.

Bronx Pediatric Society

President: Dr. Edward L. Benjamin, 2021 Grand Concourse, New York.
Secretary: Dr. Harry M. Schapiro, 1882 Grand Concourse.
Place: Concourse Plaza, 161st St. and Grand Concourse. Time: Every Second Wednesday of each month, except June, July, August and September.

Brooklyn Pediatric Society

President Dr. Carl H. Laws, 12 Pierrepont St., Brooklyn.
Secretary: Dr. Lewis A. Koch, 62 Pierrepont St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

Central New York Pediatric Club

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Fourth Thursday of October, January and April.

Chicago Pediatric Society

President: Dr. Charles Schott, 48 W. Division St., Chicago.
Secretary: Dr. Bert I. Beverly, 8 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

Dallas County Pediatric Society

President: Dr. P. E. Luecke, 4105 Live Oak St., Dallas, Texas.
Secretary: Dr. R. S. Usry, 1835 Garrett Ave., Dallas, Texas.
Place: Bradford Memorial Hospital. Time: 1 p. m., first and third Saturdays of each month.

Detroit Pediatric Society

President: Dr. Bernard Bernbaum, 5057 Woodward Ave., Detroit.
Secretary: Dr. Sarah S. Schooten, 5057 Woodward Ave., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N. E., Atlanta.
Secretary: Dr. T. I. Willingham, 478 Peachtree St., N. E., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: Second Thursday of each month from October to April, 8 p. m.

Intermountain Pediatric Society

President: Dr. Helmina Jeidell, 11 Exchange Pl., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Edwin R. Murphy, 9 Exchange Pl., Salt Lake City, Utah.
Place: Salt Lake City, General Hospital. Time: First Thursday of each month, 8 p. m.

Kansas City (Missouri) Pediatric Society

President: Dr. Hugh L. Dwyer, 406 W. 34th St., Kansas City.
Secretary: Dr. John Aull, 906 Grand Ave., Kansas City.

Memphis Pediatric Society

President: Dr. R. B. McCormick, 1074 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. W. D. Mims, 1024 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

Milwaukee Pediatric Society

President: Dr. Karl E. Kassowitz, 709 N. 11th St., Milwaukee.
Secretary: Dr. John H. Reynolds, 747 N. 27th St., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

New England Pediatric Society

President: Dr. Thomas A. Foster, 131 State St., Portland, Maine.
Secretary: Dr. Gerald Hoeffel, 319 Longwood Ave., Boston.
Place: Boston Medical Library. Time: Four meetings a year, occurring from September to May.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Martha Wollstein, Babies' Hospital of City of New York, New York.
Secretary: Dr. Harry Bakwin, 132 E. 71st St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northern California Pediatric Society

President: Dr. Lloyd Hardgrave, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Roland P. Seitz, 490 Post St., San Francisco.

Northwestern Pediatric Society

President: Dr. Alexander Stewart, 459 Lowry Medical Arts Bldg., St. Paul.
Secretary: Dr. E. F. Robb, 1801 Lyndale Ave., S., Minneapolis.

Philadelphia Pediatric Society

President: Dr. Joseph Stokes, 159 W. Coulter St., Philadelphia.
Secretary: Dr. James E. Bowman, 4091 Comly St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

Pittsburgh Pediatric Society

President: Dr. Henry C. Flood, 119 S. Highland Ave., Pittsburgh.
Secretary: Dr. E. R. McCluskey, Highland Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate months from October to April, inclusive.

Richmond Pediatric Society

President: Dr. F. Waring Lewis, Professional Bldg., Richmond, Va.
Secretary-Treasurer: Dr. A. S. Hurt, Jr., 2618 Grove Ave., Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: First Thursday of each month (except July, August and September).

Rochester Pediatric Society

President: Dr. H. F. Rowley, 176 S. Goodman St., Rochester, N. Y.
Secretary: Dr. J. M. Parker, 26 S. Goodman St., Rochester, N. Y.
Place: Arranged by program committee. Time: Second Friday of each month from October to May.

Rocky Mountain Pediatric Society

President: Dr. J. A. Schoonover, Republic Bldg., Denver.
Secretary: Dr. Arthur L. Esserman, Republic Bldg., Denver.
Place: Children's Hospital, Denver. Time: First Saturday of each month from September to May, inclusive, 7 p. m.

St. Louis Pediatric Society

President: Dr. Hugh McCulloch, 325 N. Euclid Ave., St. Louis.
Secretary-Treasurer: Dr. Katherine Bain, Beaumont Bldg., St. Louis.
Place: St. Louis Medical Society. Time: Third Friday of every month.

Seattle Pediatric Society

President: Dr. Albert S. McCown, 509 Olive St., Seattle.
Secretary: Dr. Frank H. Douglass, Medical-Dental Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, 7 p. m.

Southwestern Pediatric Society

President: Dr. James E. Harvey, 60 S. Lake Ave., Pasadena, Calif.
Secretary: Dr. E. Earl Moody, 3780 Wilshire Blvd., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Gustave F. Weinfeld, Moraine Hotel, Highland Park, Ill.
Secretary: Dr. John P. Parsons, 320 S. Division St., Ann Arbor, Mich.
Place: Ann Arbor Mich. Time: November, 1933.